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The Journal of Thoracic Surgery

VOL. I AUGUST, 1932 No. 6

Original Communications

CERTAIN ASPECTS OF THE DIAGNOSIS OF BRONCHOSTENOSIS*

FREDERICK T. LORD, M.D.

BOSTON, MASS.

I HAVE chosen bronchostenosis as the subject of this address on account of its frequency and importance. Examples of bronchostenosis are matters of almost daily observation in the medical wards of any large hospital. Bronchial obstruction occurs most often as a consequence of tenacious secretion complicating bronchopulmonary infections, especially in children. It is also common in connection with postoperative pulmonary complications. It occurs in about one out of every ten cases seen in the Thoracic Clinic at the Massachusetts General Hospital and in this group the principal causes in order of frequency are inflammatory tissue, bronchial tumors, foreign bodies, and encroachment on the bronchi from without.

During the eighteenth century, little attention was paid to the clinical aspects of stenosis of the air passages from causes other than the aspiration of foreign bodies. The diagnostic data available to a physician of this period may be illustrated by the following case:

On Monday, March 19, 1759, a child of seven threw a dried bean into her mouth and thought she had swallowed it. Convulsive cough and shortness of breath immediately followed. Several surgeons were called and after several attempts a fine sponge attached to the end of a flexible whale bone was passed throughout the length of the esophagus, but the child continued from time to time to cough with such violence as to excite convulsive movements of the extremities. She was able to swallow water

*President's Address. Read at the Meeting of the American Association for Thoracic Surgery, Ann Arbor, Mich., April 19, 1932.

and oil without great difficulty. After two days of suffering she was seen by Louis,[1] who found her sitting up in bed supported on her elbows with no other symptom than very labored, difficult, and rattling respiration and the expectoration of frothy material, and pointing with the finger to the trachea as the seat of the trouble. From the history and mere inspection he did not hesitate to say that the bean was in the trachea and that bronchotomy alone would save her life. The parents hesitated to accept this advice and instead administered an emetic without favorable effect. She died on the following day after several suffocative attacks, three days after the accident. At postmortem examination on laying open the trachea the bean was easily removed by means of small forceps.

The diagnosis in this case was made from the history and simple inspection. Recognition of the obstruction of deeper parts of the air passages was delayed until other methods of examination could be applied to the problem. Auenbrugger's discovery,[2] published in 1761, of the change in the percussion note in consequence of abnormal conditions of the lung and pleura was a necessary preliminary, but it was not until the appearance of Laennec's treatise on auscultation[3] in 1819 that definite progress was made.

Development of our knowledge of the results of bronchostenosis and the aerodynamics involved may be sketched as follows:

Pulmonary emphysema as a consequence of valvular obstruction was recognized by Laennec who ascribed it to imprisonment of inspired air behind an obstruction, assuming in explanation a relative weakness of the muscles of expiration. Inspiratory widening and expiratory narrowing of the air passages as a cause was not appreciated until the bronchoscopic observations of Ingals[4] in 1905 and of Gottstein[5] in 1907. "Collateral circulation" of air as a safeguard against it in other than lobar obstruction was demonstrated experimentally in animals by Lindskog and Van Allen[6] in 1932.

Recognition of atelectasis as a consequence of bronchostenosis proved a more difficult problem, because of the readiness with which it was confused with pneumonia. There is nothing in the writings of Laennec to indicate that he appreciated its nature, frequency, or importance. The acquired form of atelectasis was described in 1844 by Legendre and Bailly,[7] who noted among other favoring circumstances bronchial obstruction by thick and abundant mucus, but the full importance of bronchial obstruction in this connection was not appreciated until the experimental production of obstruction atelectasis in animals in 1845 by Mendelsohn.[8]

Fuchs[9] in 1849 suggested in explanation of the mechanism that the air was received into the circulating blood and this was established in 1878 by the remarkable experiments of Lichtheim.[10] Diffusion in conse-

quence of lack of equilibrium between the gases in the affected region and the venous blood is thought by Henderson[11] and by Coryllos and Birnbaum[12] to be the underlying principle. Deflation in consequence of ball-valve action of the plug was suggested in 1850 by Gairdner,[13] and the suddenness with which obstruction atelectasis develops in certain cases lends support to the view that air may at times be forced by a plug during expiration and not renewed during inspiration. Bronchoscopic observation of inspiratory closure and expiratory release of a bronchial obstruction seemed to confirm this theory, but Lindskog and Van Allen's[6] findings suggest that exhaustion of air in this way is impossible under the natural conditions of bronchial obstruction.

In the discussion of the clinical recognition of bronchostenosis, time permits a consideration only of the more essential aspects of the symptoms, physical signs, and roentgen-ray examination.

The symptoms of bronchial obstruction are more striking and typical the more sudden the obstruction and the larger the occluded passage. They are of special diagnostic importance in the case of aspirated foreign bodies following which there may be convulsive cough and threatening suffocation. The occurrence of primary and secondary symptoms with an intervening period of comparative latency long recognized and emphasized anew by Gross[14] in 1854 leaves little room for doubt as to foreign body inhalation.

In cases in which bronchostenosis complicates bronchopulmonary infection and is due to obstruction by tenacious secretion as in broncho- or lobar pneumonia, there are usually no symptoms which can with certainty be ascribed to the resulting atelectasis. If a large bronchus is plugged, there may be a greater degree of dyspnea than is to be expected from the pulmonary infection alone.

In postoperative massive collapse the relation of which to bronchial obstruction was suggested by Elliott and Dingley[15] in 1914, and established bronchoscopically by Lee and Tucker[16] in 1925, the symptom complex is at times sufficiently characteristic to suggest the diagnosis. With bronchostenosis from malignant bronchial tumor the evolution and grouping of symptoms may also be sufficiently suggestive to be of diagnostic importance.[17] A persistent cough following a "cold" may be the first manifestation. The cough is likely at first to be dry and after some time accompanied by scanty mucoid or mucopurulent and not infrequently blood-streaked or bloody sputum. At times hemoptysis is the first symptom, and recurring hemoptyses may occur at long intervals with complete freedom from any significant symptoms in the interval. The time of occurrence and degree of dyspnea depend on the size of the involved bronchus and the degree of obstruction. If the affected passage is large, dyspnea may be an early symptom. Wheezing is at times noted by the patient and may be present only when lying on the affected side.

The evidence obtained from the history is not distinctive of bronchostenosis. It is likely to be most suggestive of the diagnosis in the case of foreign body inhalation. In a large proportion of patients with bronchial obstruction there are no symptoms which can with certainty be ascribed to the obstruction.

Regarding physical signs, the essential findings with obstruction emphysema were described by Laennec. With emphysema confined to one lung or greater in one than the other, he noted enlargement of the affected side, widening of the intercostal spaces, a very clear percussion sound and diminished or absent respiratory murmur. As the valvular action of bronchial obstruction is discussed in this connection, he must, I think, be credited with the discovery. The full complex of resonance, hyperresonance or tympany on percussion, diminished or absent vesicular breathing, diminished or absent voice (without egophony), and diminished or absent whisper and tactile fremitus, as we understand it today, adds little of importance to his observations. Attention should be paid to the quality of the breathing. Absence of bronchial quality is an important feature.

There is only one other condition with a similar complex of physical signs, and that is pneumothorax. The combination of diminished respiratory murmur and resonance on percussion with amphoric breathing, metallic tinkle, and at times succussion splash was thought sufficient by Laennec to distinguish pneumothorax from emphysema due to bronchial obstruction. Roentgen-ray investigation has shown, however, that physical signs alone do not serve to distinguish a small pneumothorax from obstruction emphysema.

The essential complex of physical signs with obstruction atelectasis, dullness, and diminished breathing was noted in 1844 by Legendre and Bailly,[7] but without reference to the importance of bronchial obstruction in its production. Dullness and absent breathing in consequence of complete bronchial occlusion was noted in 1849 by Fuchs.[9] Gairdner,[18] in 1850, appreciated the rapidity with which the signs may develop and disappear following relief of the obstruction.

Concerning the signs with partial bronchial obstruction, a remarkable feature is the extent to which at times a bronchus may be occluded without corresponding evidence on physical examination. Laennec[19] in 1824 noted that almost complete plugging of the left primary bronchus by a polypus-like concretion, with not more than half a line of space for the passage of air, failed to impede respiration on the affected side. The diagnostic importance of diminished respiratory motion on the affected side with a foreign body in a bronchus was especially emphasized by Riegel[20] in 1875. Brünings[21] in 1912 noted under such circumstances that there may be no other finding and confirmed the observation by

artificially occluding a main stem bronchus to about one-fourth of its lumen. Even with almost complete occlusion there may be little else of significance. In one of our cases with a malignant tumor obstructing a left primary bronchus to about five-sixths of its extent the only physical signs were diminished motion during forced inspiration and diminished respiratory murmur during quiet breathing. Sibilant and sonorous râles in consequence of partial obstruction have been appreciated since the time of Laennec. Jackson[22] in 1921 called attention to what he termed an "asthmatoid wheeze" audible at the open mouth from narrowing of a large bronchus. Such other signs with partial bronchial obstruction, as dullness, diminished voice, whisper, and tactile fremitus have long been appreciated and are due to the trapping of secretion behind the plug and inflammatory changes with or without atelectasis in the affected region.

The complex of signs with bronchial obstruction and consequent atelectasis, retained secretion or other pathologic condition in the affected region; i. e., dullness, diminished or absent vesicular breathing, diminished voice without egophony, and diminished or absent whisper and tactile fremitus is of great diagnostic value. It is not distinctive and is found over slowly growing tumors, cysts, and the elevated diaphragm. Accommodation of the lung to slow encroachment from within or without eliminates retraction or compression atelectasis and the resulting signs of consolidation with open bronchus. Over large lung abscesses containing fluid which communicates imperfectly or not at all with the bronchus the complex of signs may also be the same as with atelectasis with closed bronchus. Obstruction atelectasis is one of few bronchopulmonary conditions in which physical examination may exceed in value the roentgen-ray in diagnosis. Though the latter is important in establishing the presence of atelectasis, it cannot be expected alone to furnish evidence sufficient to judge whether the bronchus leading to the affected region is open or closed, if the cause of the obstruction is nonopaque to the x-ray. In cases in which total collapse results from bronchial obstruction and the atelectasis persists after relief of the occlusion the signs are those of consolidation with open bronchus. Physical signs are of greater value than the x-ray examination in the decision regarding the desirability of bronchoscopy or its repetition, and by attention thereto patients may at times be spared a needless ordeal.

It is important to appreciate that with bronchostenosis and atelectasis the signs are the same with or without a complicating effusion of fluid in the space between the collapsed lung and the chest wall and the difficulty of detecting the presence of such an effusion is in consequence much increased.[23]

Application of the roentgen-ray is the most important recent advance

in the recognition of bronchostenosis and its consequences. In addition to the discovery of opaque foreign bodies there are important findings secondary to the obstruction.

Distinctive roentgen-ray findings with valvular bronchial obstruction and consequent overdistention of the lung were described in 1911 by Iglauer[24] and elaborated in 1920 by Manges.[25] Increased transparency of the affected lung, depression and partial fixation of the corresponding leaflet of the diaphragm, displacement of the heart and mediastinum away from the lesion, and increased excursion of the diaphragm on the unaffected side are significant features. The signs are accentuated at the end of expiration and complete investigation should include films at full inspiration and the end of expiration. The roentgen-ray findings with obstruction emphysema are readily differentiated from those with pneumothorax and this method thus exceeds in value the results of physical examination.

The roentgen-ray findings with obstruction atelectasis comprise changes secondary to the deflation of the affected part of the lung and those due to the atelectatic area itself. Of the secondary changes inspiratory dislocation of the heart and mediastinum toward the affected side with unilateral bronchostenosis was noted in 1899 by Jacobson[26] and by Holzknecht[27] in the same year. Narrowing of the lung field and intercostal spaces, increased downward inclination of the ribs, elevation and diminished motion of the leaflet of the diaphragm on and displacement of the heart toward the affected side and return to normal after removal of the obstruction were soon appreciated.

With respect to the atelectatic area itself, increased density in the affected region and its rapid disappearance after removal of a foreign body was also noted early in the course of x-ray studies. But in spite of rapid progress in the interpretation of the roentgen-ray findings, the diagnostic significance of the shadows produced by lobar atelectasis was not appreciated until the publications by Manges[28] in 1924, Golden[29] and Jackson and Lee[30] and Churchill[31] in 1925 and Churchill and Holmes[32] in 1927. In consequence of these observations, collapse of a lower lobe may be recognized by the presence of a dense, homogeneous shadow in the cardiophrenic angle with sharply defined outer border declining from the root downward and outward to the diaphragm and collapse of an upper lobe as a dense shadow with sharply defined concave lower margin, extending from the root upward and outward to the lateral chest wall. Collapse of the middle lobe in a number of proved cases in our experience has presented a dense shadow in the mesial aspect of the right, lower lung field with sharply defined nearly horizontal upper and vertical outer border, as reported in 1930.[33]

The roentgen-ray in the diagnosis of obstruction atelectasis is, like other methods of investigation, subject to limitations. Inspiratory dis-

location of the heart and mediastinum occurs also with pneumonia. Contraction of the side with displacement of mobile structures toward the affected region may follow in the course of inflammatory thickening of the pleura or lung with resulting fibrosis and contraction. The characteristic shadow of lobar atelectasis with or without its secondary manifestations is, however, not likely to be confused with other conditions.

Deductions from roentgen-ray findings regarding the condition of the bronchi in collapsed areas are of uncertain value. The lung may persist in a collapsed state after relief of the obstruction. Bronchostenosis may be secondary to bronchopulmonary suppuration without x-ray evidence of atelectasis.

In conclusion let me say that progress in the clinical recognition of bronchostenosis is due to an understanding of its consequences, their mode of production and to advances in methods of investigation. Evidence of great value in diagnosis may be obtained from the history, physical signs and roentgen-ray examination. None of the three can be safely neglected. Physical signs are of chief importance in obstruction atelectasis and roentgen-ray examination in obstruction emphysema.

For the determination of the nature of the obstruction other means must of course be considered. Perfection of bronchoscopic methods by Killian[34] and Gottstein[5] in Germany, and by Jackson[35] in America, has gone far toward surmounting the difficulties connected with this aspect of the problem.

REFERENCES

1. Louis: Second Mémoire sur le Bronchotomie où l'on traite des corps étrangers de la trachée artére, Mém. Acad. roy. de Chir. **4**: 513, 1768.
2. Auenbrugger: Inventum Novum ex Percussione thoracis humani, ut signo, abstrusos interni pectoris morbos detegendi (published in 1761).
3. Laennec: De l'auscultation médiate, ou Traité du diagnostic des maladies des poumons et du coeur, 1819.
4. Ingals: Respiratory Movements of the Bronchial Tubes, J. A. M. A. **45**: 1302-5, 1905.
5. Gottstein: Diagnose und Therapie der Fremdkörper in den unteren Luftwegen, Mitt. a. d. Grenzgeb. d. Med. u. Chir. Jena, 3er Suppl. Gedenkband f. J. von Mikulicz, 1907.
6. Lindskog and Van Allen: Arch. Surg. **24**: 204-230, 1932.
7. Legendre and Bailly: Arch. gén. de méd. jour. complémentaire des sc. méd., 1844, 4 ser. iv.
8. Mendelsohn: Der Mechanismus der Respiration und Circulation, etc., Berlin, 1845.
9. Fuchs: Die Bronchitis der Kinder, Leipzig, 1849.
10. Lichtheim: Arch. f. exper. Path. u. Pharmakol. **10**: 54, 1878-79.
11. Henderson: J. A. M. A. **95**: 572, 1930.
12. Coryllos and Birnbaum: Arch. Surg. **21**: No. 6, 1930.
13. Gairdner: Monthly J. M. Sc. **11**: 242, 1850.
14. Gross: Foreign Bodies in the Air Passages, 1854, Blanchard & Lee.
15. Elliott and Dingley: Lancet **1**: 1305, 1914.
16. Lee and Tucker: Tr. Coll. Phys. Philadelphia **47**: 231, 1925.
17. Lord: New England J. Med. April 14, 1927; also New England J. Med. **206**: 1471, 1932.
18. Gairdner: Monthly J. M. Sc. **11**: 122, 183, 230, 1850.
19. Laennec: Rev. méd. March, 1824, p. 384.

20. Riegel: ,Handbuch der Krankheiten des Respirations—Apparatus 1, Zweite Hälfte, Leipzig, 1875.
21. Brünings: Deutsche med. Wchnschr. 1912, No. 20, p. 929.
22. Jackson: Am. J. M. Sc. May, 1921, p. 625.
23. Lord: Med. Clin. North America **15**: 869, 1932.
24. Iglauer: Ohio State M. J. April, 1911; Lancet Clinic CVII, June 1, 1912.
25. Manges: Am. J. Roentgenol. **7**: No. 6, 1920; ibid. **9**: No. 5, 288-304, 1922.
26. Jacobson: Festschrift f. J. Lazarus, 1899.
27. Holzknecht: Wien. klin. Rundschau, 1899.
28. Manges: Am. J. Roentgenol. N. Y. **11**: 517, 1924.
29. Golden: Am. J. Roentgenol. N. Y. **13**: 21, 1925.
30. Jackson and Lee: Ann. Surg. **82**: 364, 1925.
31. Churchill: Arch. Surg. **11**: 489, 1925.
32. Churchill and Holmes: Arch. Surg. **14**: 1093, 1927.
33. Lord: Am. Coll. of Phys., Minn., Feb. 10, 1930; also Med. Clin. North America, January, 1932.
34. Killian, Kallofrath: Münch. med. Wchnschr. 1897, p. 1038; ibid. 1899; Deutsche med. Wchnschr. Aug. 31, 1921.
35. Jackson: Bronchoscopy and Esophagoscopy, W. B. Saunders, 1922.

THE PRESENT STATUS OF ONE HUNDRED PNEUMOTHORAX PATIENTS AFTER FROM ONE TO EIGHTEEN YEARS' EXPANSION OF THE LUNG*

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MUCH has been written about the treatment of pulmonary tuberculosis by artificial pneumothorax. A wealth of statistics is available showing the effects of this form of treatment upon the symptoms and the course of the disease. The indications and contraindications, the type of the collapse, the complications, and other innumerable features have been studied and recorded from every conceivable angle. Surprisingly little, however, has been written about the reexpanded lung. Does artificial pneumothorax effectually cure the tuberculosis in the collapsed lung, or is there danger after reexpansion of relapse of the

TABLE I
TIME ELAPSED SINCE PNEUMOTHORAX WAS STOPPED

TIME ELAPSED	NO. OF CASES	NO. OF DEATHS
Over 1 year	17	9
Over 2 years	22	4
Over 3 years	10	3
Over 4 years	10	1
Over 5 years	11	2
Over 6 years	10	2
Over 7 years	7	0
Over 8 years	5	1
Over 10 years	2	0
Over 11 years	3	2
Over 12 years	1	0
Over 14 years	2	0
Over 17 years	4	0
Over 18 years	1	0

disease? How long should the pneumothorax treatment be maintained in order to be reasonably sure of a permanent arrest of the disease? Is the patient permanently handicapped after the pneumothorax treatment is stopped? These and other questions present themselves when the cessation of artificial pneumothorax treatment is considered.

In an attempt to answer such questions the present status of 105 former pneumothorax patients has been studied. One-half of the cases is taken from the records of Trudeau Sanatorium and the New York State Hospital at Ray Brook to whose staffs I am indebted for assistance. The other half is from private practice. In this series of patients col-

*Read at the Fifteenth Annual Meeting of the American Association for Thoracic Surgery at Ann Arbor, Michigan, April 18, 19, 20, 1932.

lapse therapy has been discontinued for at least one year. Table I shows the number of cases in each year period since pneumothorax was stopped up to January, 1932, or to the year in which the patient died.

The average length of reexpansion in this series is a little over five years. It is noteworthy that the majority of the fatal cases occurred within three years after the cessation of pneumothorax treatment and that between the fourth and eighteenth year period only 8 deaths occurred among 56 individuals. Evidently, the longer the patient "carries on" after reexpansion the less likelihood of relapse.

When the term "reexpanded lung" is used, it must be borne in mind that the formerly collapsed lung does not reexpand to its original size. Fibrosis, atelectasis, and pleural thickening cause a shrinkage in lung volume, and in a few instances there is but little actual reexpansion of lung tissue, the pleural cavity being obliterated by a displacement of the collapsed lung and mediastinum and by compensatory changes in the chest wall and other thoracic organs.

CLASSIFICATION OF CASES

The majority of the cases were in the far advanced group, and all except 25 had more or less disease in the opposite lung. Seventy-three had definite cavity formation of varying size. There were 13 patients whose lungs were collapsed because of hemoptysis in whom no cavity could be demonstrated. The remaining cases were for the most part composed of the progressive toxic type. The presence of persistent cavity is the great and impelling indication for collapse therapy, and unless the cavity can be collapsed the success of treatment is greatly diminished and the prognosis correspondingly poor.

All of this group of patients were observed for many months before pneumothorax was instituted. Conservatism in treatment was the rule in their day. During the past few years the tendency is to start collapse therapy in earlier cases, and after shorter periods of observation.

IN REGARD TO TREATMENT

The type of collapse has little to do with the end-results provided the cavity, if present, is obliterated and the sputum rendered negative for tubercle bacilli. A pulmonary collapse of from 40 to 50 per cent and numerous adhesions present may nevertheless produce brilliant results, while a 90 per cent collapse but with a patent apical cavity and positive sputum is doomed to eventual failure, unless surgical measures are employed to collapse the cavity.

In 24 cases the sputum remained positive during treatment. In this group were 21 cavity cases, and of these there were 18 patients whose cavities were but partially collapsed. Of these positive sputum cases none is working; 10 or 42 per cent are "curing," and 14 or 58 per cent

are dead. Out of 98 positive sputum cases, there are 75 patients whose sputum became negative after artificial pneumothorax was instituted. In this group all cavities when present were collapsed.

It is interesting to note that 66 of these patients had a left-sided pneumothorax, and that only 39 were on the right side.

A serous effusion developed in 74 cases, but these were mostly of small amount. Twelve of the effusions became purulent, and one was a mixed infection.

TERMINATION OF TREATMENT

Dumerest[1] is of the opinion that a successful pneumothorax should be continued indefinitely, while Rist[2] believes that the minimum duration of treatment should be five years. In about half of this series in which the method of termination was recorded the question automatically settled itself by the obliteration of the pneumothorax space either by the development of effusion and the gradual reexpansion of the lung beneath the fluid; or by the reexpansion of the lung in spite of frequent high pressure refills; or by the shifting of the collapsed lung and mediastinum into the pneumothorax side with eventual blocking of the air space. The average length of time from the beginning of treatment to its involuntary termination was slightly over two years.

The average duration of the pneumothorax in those of this series who are recorded as voluntarily abandoning treatment was somewhat over two and one-half years. One patient continued his pneumothorax refills for fourteen years! The average duration of treatment for the 105 patients was 2.6 years.

It is worthy of comment that of 54 patients who are working, one half lost their pneumothorax involuntarily, while the other half stopped treatments as a matter of choice. When a patient with a satisfactory collapse that has been maintained for at least two years, and whose sputum has been consistently negative, begins to lose his pneumothorax space in spite of treatment the final result should be looked upon with equanimity, not with fear.

END-RESULTS

Of the 105 patients whose lungs have been reexpanded for periods of one to eighteen years, 81 are alive and 24 are dead. Of the 81 who are alive 62 (76+ per cent) are working or leading normal lives, and 19 (23+ per cent) are still "curing," although 5 of these report themselves as "well." Amberson[3] in a study of 165 reexpanded cases arrived at practically the same figures.

How do these results compare with a similar group not treated by artificial pneumothorax? It is difficult to make true comparisons. If we take the so-called "control" group, those in whom artificial pneumothorax was attempted but without success, we find a mortality of about

60 per cent at the end of two years. But this is like comparing the winners of a race with the losers. It is not a just comparison.

The X-Ray Department of Trudeau Sanatorium has furnished me with a group of 154 cavity cases classified as moderately or far advanced. They had sanatorium treatment, but no form of collapse therapy was employed. Sixty-nine per cent were dead at the end of a five-year period. The best control group would be a series of cavity cases whose cavities closed without surgical aid, and see how these patients have done from one to eighteen years after the closure of the cavity. But such a group is not available for study at the present time.

ANALYSIS OF RESULTS

In a broad survey of these 105 patients under discussion we find that those who are living and well obtained a satisfactory collapse of their cavity and the sputum became negative for tubercle bacilli; among those who are still curing, pneumothorax failed to close the cavity in half of the cavity cases, or there was progression of disease in the other lung, or the patients underwent further surgical procedures from which they have not yet recovered. Of those who died, pneumothorax failed to produce a satisfactory collapse of the lung in the majority of cases, cavities remained patent, or there was an extension of disease in the contralateral lung. Nine of the 24 fatal cases died between the first and second year after the cessation of treatment. Four patients died of disease other than their pulmonary tuberculosis.

In regard to the reactivation of the disease after the collapsed lung has reexpanded my observations are limited to 50 cases seen in private practice. There were 38 patients who returned to work and of these only 3 had a relapse in the reexpanded lung. A fourth died of meningitis three years after reexpansion, while a fifth died suddenly after three years of work, of what was reported as pneumonia.

In one of the relapsed cases the former cavity reappeared three years after the cessation of the pneumothorax. This patient has since had thoracoplasty. In the second patient an acute tuberculous process with cavitation developed in the opposite lung. This lung was collapsed and in the meantime a cavity appeared in the formerly collapsed lung, but not at the site of the old cavity. This patient is still curing. In the third case the cavity broke down seven years after the cessation of pneumothorax, during which time the patient had worked steadily. The lung was recollapsed, but the cavity never closed completely. Empyema developed, and the patient is still on the sick list.

In the sanatorium group of 55 cases there were 34 who returned to work. Five of these relapsed, of whom 3 died five, eight, and eleven years respectively after pneumothorax. Two are curing. The records do not show the cause of the relapses, although curiously none of these

5 patients had demonstrable cavity before pneumothorax was begun. If we assume that reactivity occurred in the reexpanded lung, we have 10 patients who relapsed out of the 72 working individuals. It is noteworthy that these relapses occurred from three to eleven years after pneumothorax was stopped! In 51 cases of reexpanded lung reported by Rist[4] in 1927, only 2, at that time, had relapses in the formerly collapsed lung. These figures seem to show that a breaking down of the reexpanded lung is the exception rather than the rule.

THE REEXPANDED LUNG

A fear has been engendered that the reexpanded lung is a most delicate organ and that a slight strain will break it down; that the suction upon the reexpanding lung by the pleural vacuum is so great that healed areas will be torn open and active disease again appear; that the patient suffers from pain and dyspnea during the process of reexpansion; that he may always have a pneumothorax due to the nonabsorption of the pleural air; that marked distortion of the chest and thoracic organs takes place as a result of the compensatory changes when the pneumothorax is stopped. Because of these dangers some advocate the pneumothorax be continued indefinitely, or that oleothorax, or thoracoplasty be resorted to.

In general, my experience with the reexpanded lung has been quite the opposite. Of course there are patients who insist on maintaining the pneumothorax for years and in such cases I do not argue, especially if the lung before collapse contained a very large cavity. There are others with poor collapses who may be helped by oleothorax. Another group requires thoracoplasty for an effectual closure of the cavity. But in the series of cases under discussion we are dealing with those who have, as it were, survived the fire and sword. Given a case with a moderate sized cavity who obtains an effective collapse and negative sputum for a period of two and a half years, I see no reason why the lung should not be allowed to reexpand gradually. The figures presented do not show that the process of reexpansion caused a flare-up of the disease.

A few of the patients were a bit uncomfortable when the lung was reexpanding if the pleural pressures became too low, but their symptoms were quickly relieved by the introduction of air. As soon as the lung was "out" against the chest wall, symptoms disappeared. In only one case was there real dyspnea which developed more and more as the so-called reexpanded lung continued to contract, and the heart was displaced to the left. Finally, a thoracoplasty was done to relieve the pull upon the heart and mediastinum and opposite lung. Relief was obtained, but the patient is still somewhat short of breath.

In none of this series did a pneumothorax persist after the cessation of treatment.

Out of this group there are 3 or 4 who have a marked thoracic deformity, that is a flattened, narrowed, more or less immobile chest on the affected side. The majority of patients have surprisingly little outward deformity, although sustaining some cardiac displacement and pleural thickening as shown by the roentgen ray. These patients have fair expansion, and in many patients the mobility of the affected side is nearly if not entirely equal to the other side.

Surgical measures played little part in this series. Two had thoracoplasty, as already mentioned. Only 5 phrenicectomies were performed, two as a preliminary measure to reexpansion, and three in an attempt to close cavities.

The arguments in favor of a phrenicectomy during the reexpansion process are logical, but in the group under discussion the majority of cases got along very well without it.

CONCLUSIONS

A review of 105 reexpanded lungs from one to eighteen years after pneumothorax therapy shows:

1. That 81 are alive and 24 dead. Of those who are alive 62 (76 per cent) are working, and 19 (24 per cent) are "curing."

2. That a successful collapse will effectually heal the lung in the majority of cases.

3. That during the process of reexpansion and afterward reactivity of the disease is the exception rather than the rule in those who have had a satisfactory collapse.

4. That an effective pneumothorax of two years' duration, and a negative sputum, for at least a year, give reasonable assurance that the lung may be expanded with safety.

5. That those patients with closed cavities and negative sputum, who unintentionally lose their collapse at the end of two years, have no more to fear a reactivity of the lesion than those who can continue treatment for much longer periods.

REFERENCES

1. Dumerest, and Brette: La Pratique du Pneumothorax, Masson et Cie, 1929.
2. Rist, E.: Quand faut-il interrompre le pneumothorax therapeutique? Rev. de la tuberculose **9**: 915, 1930.
3. Amberson, J. B., Jr.: The Indications for and the Results of Artificial Pneumothorax Treatment in Pulmonary Tuberculosis, Ann. Int. Med. **4**: 343, 1930.
4. Rist, E.: Results of Artificial Pneumothorax in Pulmonary Tuberculosis, Am. Rev. Tubere. **15**: 294, 1927.

BILATERAL PARTIAL THORACOPLASTY FOR BILATERAL PULMONARY TUBERCULOSIS*

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BILATERAL surgical collapse of a portion of the chest walls offers a new aid to certain individuals in that group of patients who are suffering with tuberculosis of both lungs. The rationale for such procedures is, as in unilateral surgical procedures, to permit and aid collapse of the diseased portions of the lung and to maintain an effectual rest of the tuberculous lesion. Bilateral partial thoracoplasty will permit the consummation of these aims in a certain group of patients wherein the other bilateral surgical procedures are of no avail.

Bilateral partial thoracoplasty is the logical continuation and outgrowth of these surgical procedures.

The first of these to be employed in the treatment of bilateral tuberculosis was bilateral artificial pneumothorax. This was introduced by Foralini in 1911,[1] who advocated the successive introduction of the pneumothorax into the second side after allowing the original collapsed lung to reexpand. Ascoli,[2] in 1912, advocated the use of bilateral simultaneous pneumothorax. He introduced air into both halves of the chest at a rather long interval but the first pneumothorax was maintained after and during the second. Both lungs were maintained in partial collapse over a period of time.

Livierato,[3] in 1914, reported a series of patients with simultaneous bilateral pneumothorax with an explanation for the beneficial results in slight compression of both lungs. He believed that there was a diminution of the blood flow through the diseased portion of the lung and that there was also a decrease of lymph flow from the lung to the parietal pleura. Thus he believed there was a damming up of toxic material in the lesion in the lung and a consequent diminution of toxins in the blood stream with its resultant benefits to the individual. Bezancon and Jacquelin,[4] Coulaud,[5] Samson,[6] Kinsella and Mattill,[7] Maendl,[8] Andre and De Loose[9] and many others reported series of patients in which the results of bilateral pneumothorax proved the treatment to be safe, comfortable and beneficial. Rist found that a reduction of the vital capacity to 20 per cent of its original volume was compatible with life. In our surgical collapse measures we have held that no further diminution in the vital capacity should be allowed after it had reached a volume of 1000 c.c.

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Bilateral phrenic neurectomy was first reported for the treatment of bilateral tuberculosis by Curti.[10] Following him studies by Dunner,[11] Iselin[12] and others seem to establish the safety of this treatment. Bilateral phrenicectomy is a simple procedure insofar as the operation is concerned but the results must be classified as being rather radical in certain respects. As compared to bilateral partial thoracoplasty bilateral phrenic neurectomy is more extensive in its effect in the lung. Phrenic neurectomy affects that portion of the lung which is *not* involved in the tuberculous lesion as well as that portion which is involved. Partial thoracoplasty is the more selective in that its effect is largely upon the diseased portion of the lung and little, if any, effect is transferred to the respiratory function of the uninvolved portion of the lung.

Both artificial pneumothorax and phrenic neurectomy have been performed in the chest on the opposite side of which other surgical procedures had been carried out. In 1929 P. Anneuille[13] reported the first case in which an artificial pneumothorax was established in the side of the chest opposite to which a complete thoracoplasty existed. Maendl[14] reported a patient in which a Brauer type thoracoplasty was made on the left side followed by an activation of a preexisting tuberculous lesion in the right lung and the subsequent establishment of an artificial pneumothorax in the right chest. In both of these patients the bilateral procedures were well tolerated. Numerous instances are on record in which a pneumothorax has been established in one-half of a chest subsequent to or prior to removal of the phrenic nerve supply in the diaphragm for the opposite side.

The indications for the feasibility of bilateral partial thoracoplasty in patients has been established in experimental studies, as well as in clinical studies. It has seemed to us that the most basic consideration for this is the vital capacity. The experimental work of Graham and Bell[15] with bilateral open pneumothorax has a direct bearing upon all such bilateral operative measures. They found that open pneumothorax decreased the vital capacity in a very definite manner depending upon the size of the hole in the chest wall. Furthermore, they found that bilateral open pneumothorax was tolerated quite as well as unilateral pneumothorax provided the combined areas of the two openings were no greater than the area of the unilateral opening. This is the equivalent of saying that a decrease of the vital capacity in both sides of the chest would be tolerated as well as a decrease in the vital capacity in one side of the chest provided the actual decrease in both instances was the same in volume. This indicates that collapse of half of both lungs would be tolerated as well as collapse of all of one lung.

Equally significant, is the experimental work of Kinsella.[16] He made a study of bilateral upper extrapleural paravertebral thoracoplasty in dogs and monkeys. The operative procedures were carried out to simulate as nearly as possible the Wilms-Sauerbruch and Brauer procedures

in man. A multiple stage subperiosteal paravertebral costectomy was done. Segments of as many as nine ribs were removed on both sides of the apex of the chest of the dog. Subsequent regeneration produced a fixation of the chest in the collapsed position. The dogs showed no respiratory distress even during moderate exercise.

A similar experiment was carried out in a monkey. Large segments of the upper six ribs on the right side and the upper four ribs on the left side were removed. There was no embarrassment in its respiration and it was as active as its fellows. From these observations he concluded that bilateral upper paravertebral thoracoplasty could be carried out in man "without fear of gross deformity, of respiratory embarrassment due either to the extent of the collapse itself or to the fixation of the chest wall by regeneration of the ribs."

SELECTION OF CASES

The indications favorable to bilateral thoracoplasty, in addition to the presence of an adequate vital capacity, are, first, the proper anatomic distribution of the lesions within the lungs and, secondly, those factors which are ordinarily considered as favorable indications for unilateral thoracoplasty.

The most favorable anatomic distribution of the lesions within the lungs appears to be within the apices in both lungs. Perhaps the most desirable location of the lesions is in that portion of each lung which extends above the level of the fifth ribs as measured on the posterior aspect of the chest wall. Resection of segments of the upper ribs does not interfere with the respiratory function of the diaphragm and should produce the minimum restrictive effect upon the costal respiration. Furthermore more that 50 per cent of the upper four ribs can be removed without great alteration in the vital capacity and without great shock or inconvenience to the patient. An effectual collapse of the apices of the lung usually follows such unilateral apical thoracoplasties. If the diseased portions of the lungs are confined to the apices, this collapse and immobilization of them would be expected to provide a maximum of benefit to the patient.

Other indications for bilateral thoracoplasty are those which point to the possibility of withstanding the immediate effects of the operation. It is unnecessary to go into these in detail. A careful evaluation should be made of the cardiac reserve, of the renal function, of the vital capacity, the clotting time and the bleeding time and of the progress of the tuberculous lesions in the lungs. Patients in whom the tuberculous lesions of the lungs show rapid progression should not be considered as candidates for thoracoplasty.

The indications which are considered as favorable for thoracoplasty in unilateral cases are not necessarily always present in their entirety in bilateral cases. The most notable exception to this perhaps is that

the trachea need not be displaced from the midline in bilateral cases. The displacement of the trachea in unilateral disease is a measure of the presence of fibrous tissue around the lesion. Contraction of the fibrous tissue produces the displacement. If this fibrous tissue is laid down in both apices the trachea may not be displaced and yet the lesion may be of the fibrous type. It is common knowledge that the fibrous lesion responds most readily to surgical collapse therapy.

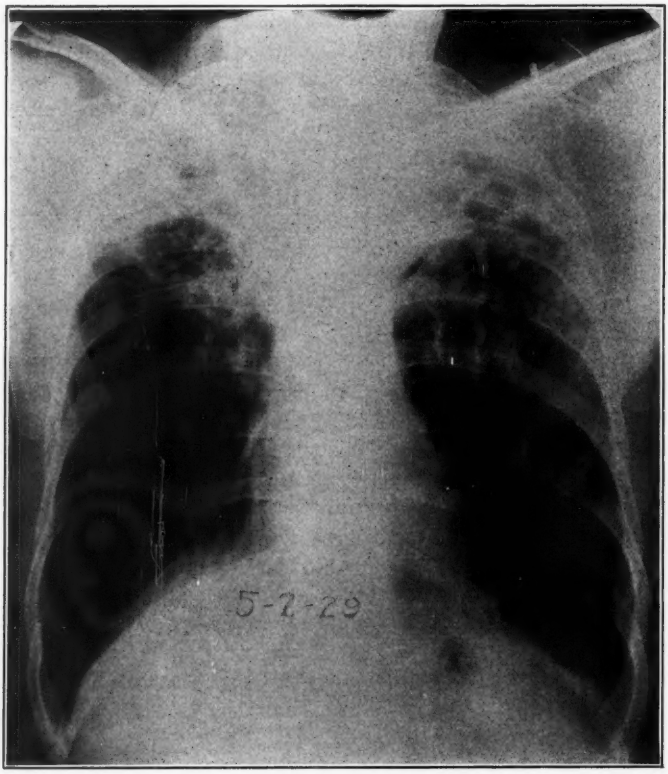

Fig. 1.—Postero-anterior roentgen plate of the chest after seven months' sanatorium care (15 years after known onset of the disease). Far advanced pulmonary tuberculosis with heavy infiltration of the right upper lobe and cavitation in the left upper lobe. Note the areas of dense fibrous tissue and the perpendicular course of the bronchi in the left lower lobe.

The following case will illustrate other features favoring bilateral partial thoracoplasty.

C. K. (Hospital No. 52783), age forty-seven, entered the Robert Koch Hospital April, 1929. He had a far advanced fibrocavernous tuberculosis of the apices of both lungs. He gave a history of the first known onset of his disease fifteen years previously. He had been able to carry on

with his work most of the time during this period. He had had a persistent cough for the past twenty years. He had had insufficient treatment for his tuberculosis at the beginning and subsequently except for the five months immediately preceding his entrance to the Robert Koch Hospital during which time he was in a sanatorium.

During the year preceding the entry to the Robert Koch Hospital he had lost twenty-four pounds. He had had repeated hemoptyses, had a

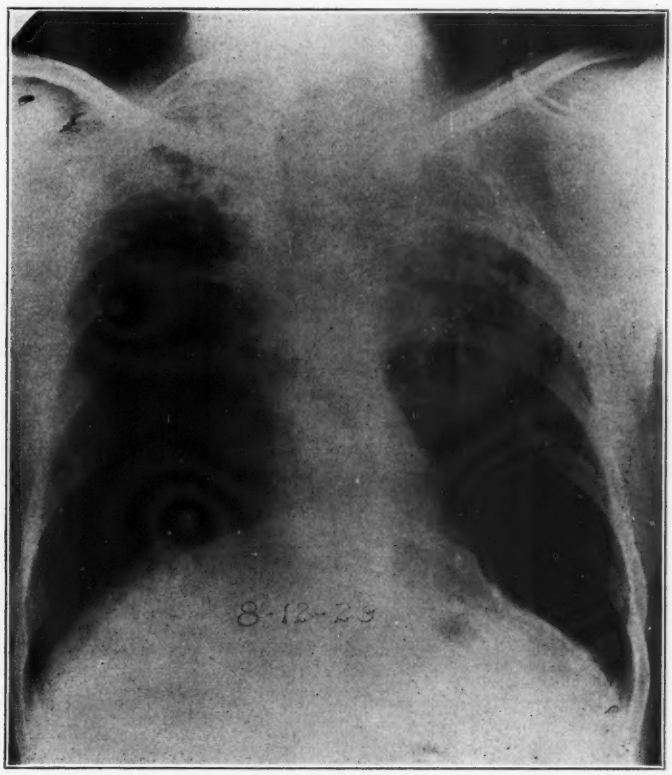

Fig. 2.—X-ray plate taken six weeks after the first (left) half of bilateral partial thoracoplasty. Compare with Fig. 1. Note that the cavities in the upper lobe of the left lung are closed and that there is a clearing up of the infiltration in the upper lobe of the unoperated right lung. Coincident with these changes there were clinical signs of improvement.

moderate daily elevation of temperature. With slight exercise there was a more marked rise in temperature.

His physical examination on entrance was typical of far advanced tuberculosis of the apices of both lungs. This was confirmed by the x-ray (Fig. 1). Sputum examinations were positive for tubercle bacilli.

He remained in bed for an additional two months making a total of seven months of sanatorium care during which time he did not improve. Pneumothorax was attempted on both sides but could not be established. It seemed evident that the ordinary sanatorium care could not be expected to bring about improvement, at least not within the next few years. He seemed to be destined to become the so-called sanatorium tramp. A unilateral thoracoplasty was considered but because of the

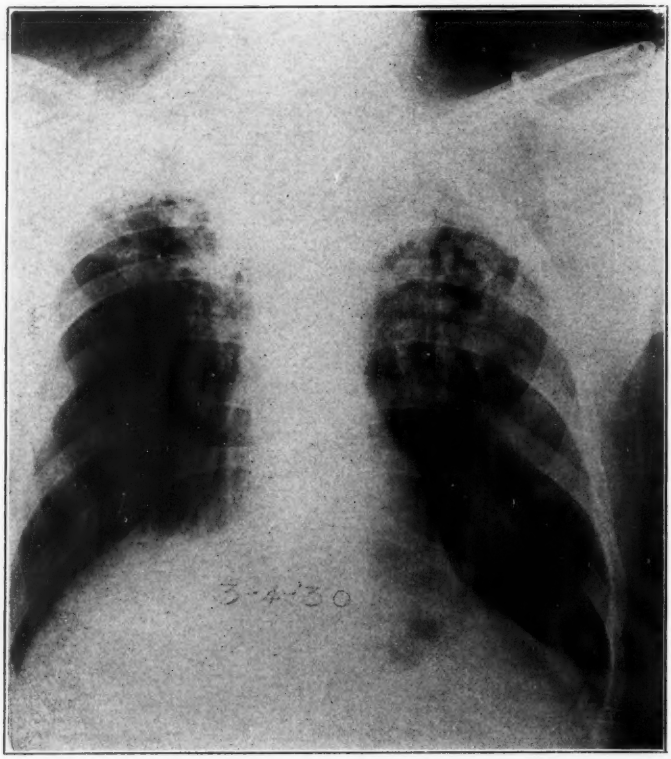

Fig. 3.—X-ray plate nine months after first operation. Note dense fibrosis in both right and left upper lobes. There are no cavities visible. The partial thoracoplasty has resulted in a good collapse of the left upper lobe.

almost equal distribution of the involvement of both lungs this appeared illogical. A bilateral phrenicectomy was considered but because of the dense consolidation about the lesions and the presence of adhesions throughout the chest this also was abandoned.

A bilateral apical thoracoplasty seemed to be the most logical procedure. Accordingly the first stage of this operation was carried out on June 29, 1929 (Fig. 2). The upper five ribs were removed from

the left chest wall beginning at the transverse processes and extending around to the midaxillary line.

The immediate postoperative course following this, the first stage of the bilateral thoracoplasty, was not unlike the usual course in similar patients in whom the lesion is confined to the one side upon which a partial thoracoplasty is carried out. There was no evidence of spread of the lesion in the lung of the unoperated side.

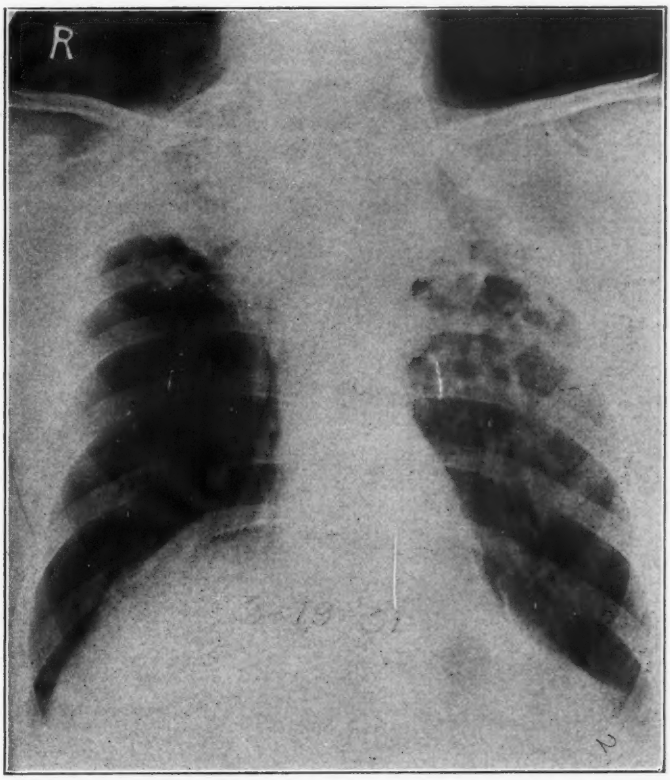

Fig. 4.—Twenty-one months after first operation on left side. Compare with Fig. 2. Note the dense fibrous tissue in the right upper lobe. There is also a dense infiltration of the left lower lobe below the collapse. There appears to be a cavity in the left lung at the level of the sixth interspace as measured posteriorly. Physical signs and the subsequent clinical course of the patient did not substantiate this.

It had been our original plan to allow about eight to twelve weeks to elapse before performing the second operation of the bilateral partial thoracoplasty upon the opposite side from the first. However, at the end of this time the signs of activity in the unoperated lung had become lessened. Coincident with this there had been an improvement in his general condition and for the first time the sputum showed no

tubercle bacilli on repeated examinations. His weight slowly increased toward the calculated normal weight. At the end of one year after the first stage of the bilateral operation he had gained to his normal weight, the sputum had decreased to an average of about 15 gms. daily, and the Schilling differential count had indicated improvement as shown in the tabulation of laboratory findings.

At this point it seemed possible that it would not be necessary to

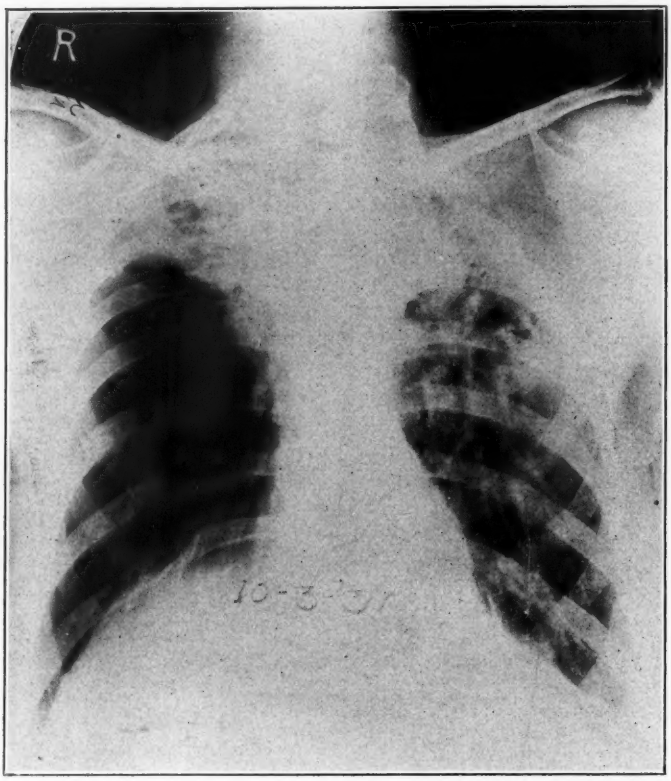

Fig. 5.—Twenty-seven months after the first operation. Compare with Figs. 3 and 4. Note that there is a definite cavity in the unoperated right upper lobe. The signs elicited by auscultation at the time this x-ray film was made, when compared with the signs obtained six months previously (Fig. 3), indicated increased activity and cavity formation in the lesion of the right upper lobe.

complete the bilateral thoracoplasty. The lesion in the opposite, unoperated, lung had become quiescent and the x-ray film showed an increase in the density of it. This was apparently due to an increase of fibrous tissue in the lesion. We had seen this phenomenon occur in other cases in which a massive lesion had existed in the side which had been operated upon and a small active lesion had existed in the oppo-

site lung. In these cases following partial apical unilateral thoracoplasty on the "worst" side we had been gratified to note almost a complete disappearance of the tuberculous lesion in the opposite lung. In fact, it was these observations of the results in cases with an extensive lesion in the apex of the lung in the side operated upon and a lesser extensive lesion in the opposite side which prompted us to consider this case with extensive lesions in both lungs for bilateral partial thoracoplasty. In

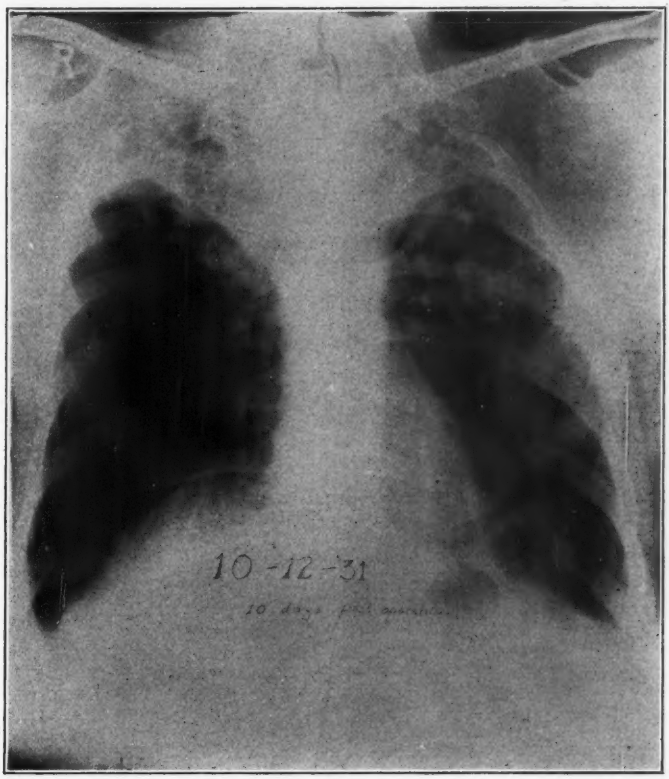

Fig. 6.—X-ray plate made ten days after completion of the second half of the bilateral apical thoracoplasty. The physical signs and clinical course of the patient indicated the effect of the collapse of the right upper lobe more clearly than did the x-ray. There were few signs of cavitation remaining, the character of the sputum had changed and there was a notable decrease in the dyspnea which the patient had experienced prior to the operation.

some of our previous cases (with hemoptysis) the "lesser" lesion in the opposite lung had been rather extensive and had shown improvement following partial thoracoplasty of the opposite side.

During the succeeding year (the second year following the first operation) his course was less favorable. The sputum again showed the pres-

ence of tubercle bacilli; there were signs of increasing activity of the lesion of the lung on the unoperated side; he lost weight gradually during the first six months of this year. He seldom had a temperature above the normal.

At the end of twenty-three months after his first operation he began to exhibit a still more unfavorable course. The sputum increased in amount and tubercle bacillus content. He lost weight rapidly. The

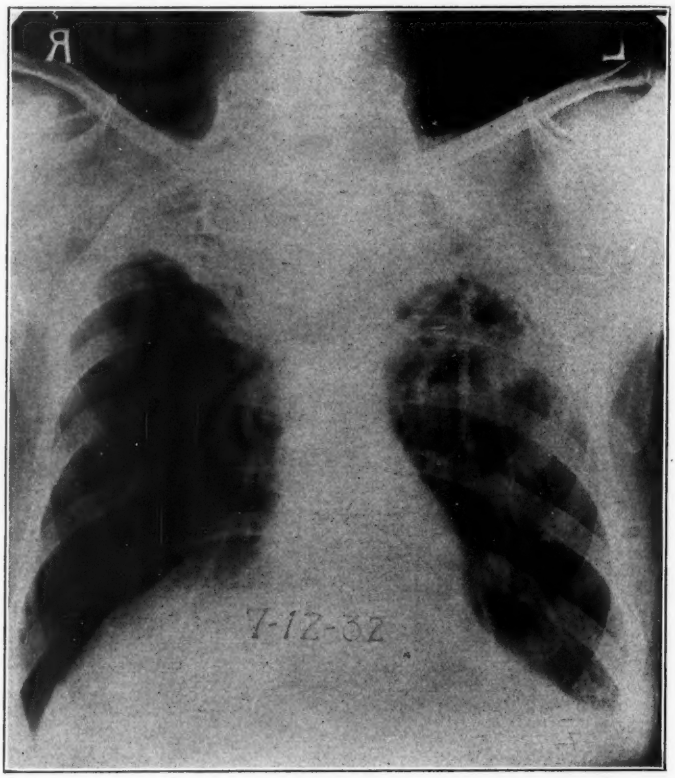

Fig. 7.—Nine months after completion of the second half of the operation. Compare with Figs. 1, 4, 5 and 6. There is less collapse of the portion of the right chest than of the left but there is sufficient collapse to close the cavity in the right upper lobe and to bring about sufficient immobilization of it to permit healing. Comparison with Fig. 5 shows the amount of collapse in the right side.

physical signs in the upper third of the right lung indicated increased activity in it.

At the end of the twenty-seventh month the second operation for the bilateral partial thoracoplasty was completed. At this operation, which was performed on October 2, 1931, portions of the four upper ribs were removed subperiosteally beginning at a point near the tip of the transverse processes of the spine and extending forward on the ribs to in-

clude about half the length of each rib. There was no notable embarrassment to the respiration or to the circulation during the performance of operation.

The immediate postoperative course after completing the bilateral partial thoracoplasty was very satisfactory. The respirations were never more than 24 per minute during the ensuing two weeks and the body temperature was never more than one degree above the normal.

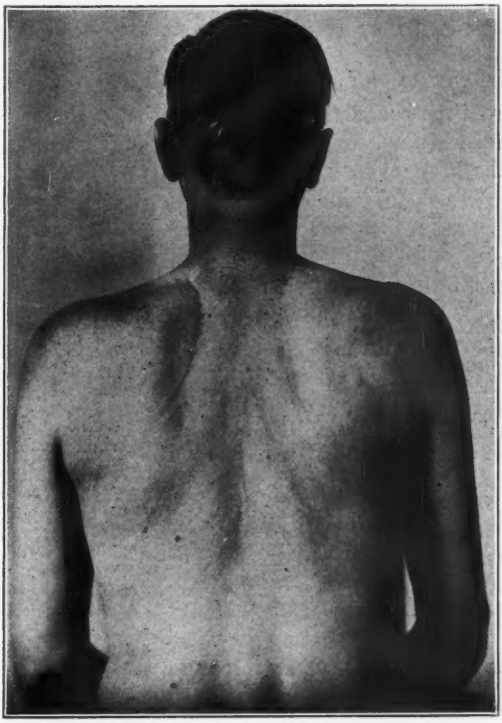

Fig. 8.—Photograph of back of patient. Note the absence of deformity. The angles between the base of neck and the top of the shoulder more nearly approach the right angle than before operation. The neck appears to be longer. There was no limitation of motion of scapula or of motion of the arms at the shoulder.

The vital capacity before this operation had been obtained as 2150 c.c. Three months after the operation it was obtained as 2075 c.c. There had been, therefore, a very slight and negligible decrease in the vital capacity. This had been anticipated since the decrease in size of the chest had been limited to the diseased portion of the lung and had affected the normal functioning portion of the lung but little.

An unexpected event was noted after the second operation. Following the first operation upon the left chest dyspnea had gradually developed. This amounted to difficulty with inspiration and, to a lesser de-

gree, difficulty with expiration. Within three or four days after the second operation had been performed the dyspnea had become much less and within the first four weeks it disappeared entirely. Its appearance and disappearance was attributed to a shifting of the trachea. It was thought that the trachea had shifted slowly toward the right following the first operation and that it had returned to the normal position after the second operation. Our x-ray films do not show this clearly. If dyspnea proves to be a frequent phenomenon it should be studied by the x-ray immediately after the introduction of lipiodol into the trachea.

Studies of the blood and sputum have shown notable evidence of improvement since the completion of the operation. The following table shows the total white cell count and Schilling differential count during his stay in the hospital.

DATE	TOTAL WHITE CELL	EOSINO-PHILES	BASO-PHILES	MYELO-CYTES	JUVE-NILE CELLS	STABS	SEG-MENTS	LYMPHO-CYTES	MONO-CYTES
4/25/29	8,700	2	0	0	1	20	52	19	6
2/26/30	7,350	2	0	0	0	16	46	27	9
9/ 6/30	5,850	0	0	0	1	11	49	28	10
4/ 2/31	7,200	3	0	0	0	14	41	34	8
10/ 9/31	6,800	1	0	0	0	21	53	13	12
2/ 4/32	7,000	5	0	0	0	9	44	33	9
6/ 7/32	7,600	2	0	0	0	12	40	36	10

All the above counts were made by the same technician.

The sputum examinations by the antiformin technic were as follows:

4/26/29	(++ Gaffky)	1/11/32	negative
4/27/29	(++)	1/12/32	negative
6/11/29	negative	1/13/32	negative
10/21/29	negative	1/14/32	negative
3/13/30	negative	4/19/32	negative
6/16/30	(+++)	4/20/32	negative
10/ 6/30	(++)	4/21/32	negative
2/17/31	(+)	4/22/32	negative
5/18/31	negative	4/23/32	negative
8/26/31	(+++)	4/24/32	negative
10/23/31	(+++)	4/25/32	negative
11/23/31	(++)	4/26/32	negative
		4/27/32	negative
		4/28/32	negative
		4/29/32	negative

The amount of sputum was but slightly decreased in quantity following the second operation, but the character changed to a clear mucoid type, not very unlike the normal saliva.

The clinical improvement following the second stage of the bilateral partial thoracoplasty was satisfactory. The patient began to gain weight immediately and within the next four months had attained his calculated normal weight. He was again placed on the sanatorium regime with graded exercises. At no time was the temperature of the body more than .2 of a degree above normal. He left the sanatorium against advice at the end of ten months after the completion of his

operation and is able to carry on at a rather exacting and moderately difficult work at the present time.

Since completing our first operation of bilateral partial thoracoplasty, we have performed the first half (unilateral) of the operation in two additional cases. These patients had extensive involvement of the apices in both lungs. In neither patient was there an increased activation in the lesion of the contralateral lung following the unilateral partial thoracoplasty. Neither of them had respiratory difficulty following the operation. Both exhibited a very satisfactory postoperative course which was unattended by alarming respiratory or circulatory symptoms.

RÉSUMÉ

The clinical experience of others is reviewed briefly with regard to bilateral artificial pneumothorax, bilateral phrenic neurectomy, combinations of unilateral phrenic neurectomy with contralateral artificial pneumothorax and these two in combination with contralateral thoracoplasty. These results in the treatment of pulmonary tuberculosis indicate the feasibility of bilateral partial thoracoplasty in patients with involvement of the upper portion of both lungs.

Theoretical considerations for the safety of the operation are found in the experimental work of Graham and Bell[15] and of Kinsella.[16] Graham and Bell showed that the vital capacity of the chest was decreased almost equally by an alteration affecting only one side of the thorax or by an equal alteration which affected both sides of the thorax. This is the equivalent of the statement which we have made in this report that, theoretically, "collapse of half of both lungs would be tolerated as well as collapse of all of one lung." Kinsella showed by experiments carried out on dogs and a monkey that bilateral upper partial thoracoplasty was well tolerated.

The report of a case is given in which a bilateral partial thoracoplasty was made. The first stage, unilateral apical partial thoracoplasty, was performed on June 29, 1929. Following this the patient showed definite improvement so that the second half, which had been planned to follow the first operation after an interval of eight to twelve weeks, was not done until twenty-seven months had elapsed. Following the completion of the bilateral partial thoracoplasty, the patient again showed improvement and returned to his work ten months later.

Two additional cases are reported in which the first half of a bilateral partial thoracoplasty has been completed. In these cases there was massive involvement of the upper portions of both lungs. In neither patient was there an increase in the activity of the process in the contralateral lung.

The observations in these three cases indicate that in certain patients with involvement of both lungs a bilateral partial thoracoplasty may be performed with safety and benefit.

REFERENCES

1. Forlanini, C.: Del pneumotorace artificiale consecutivamente bilaterale. Gaz. Med. Ital. **62**: 41, 1911.
2. Ascoli, M.: Ueber den Künstlichen Pneumothorax nach Forlanini. Deutsche Med. Wchnschr. **38**: 1782, 1912.
3. Livierato, P. E.: Indicazione del pneumotorace artificiale da un nuovo punto di vista. Riforma med. **30**: 1373, 1914.
4. Bezancon, F., and Jacquelin, A.: La Pneumothorax partiel bilateral simultane (contribution a l'etude du pneumothorax thérapeutique). Presse méd. Sept. 17, 1924.
5. Coulaud, M.: Double pneumothorax thérapeutique efficace; tolérance parfaite malgré un collapsus bilateral complet. Presse méd. **40**: 664, 1925.
6. Samson, J. W.: Pneumothorax Bilateralis. Ztschr. f. Tuberk. **44**: 177, 1925; Ztschr. f. Tuberk. **47**: 273, 1927.
7. Kinsella, T. J., and Mattill, P. M.: Bilateral Pneumothorax. Am. Rev. Tubere. **16**: 655, 1927.
8. Maendl, H.: Ueber nachtraglich doppelseitigen und gleichzeitig doppelseitigen künstlichen Pneumothorax der Lungentuberkulose. Wien. klin. Wchnschr. **40**: 347, 1927.
9. Andre and De Loose: La collapseothérapie bilatérale: essai d'explication de la plus grande frequence des perforations pulmonaires au cours du deuxième pneumothorax. Rev. Belge d. l. Tubere. **19**: 26, 1928.
10. Curti, E.: Risultati a distanza della frenicotomia bilaterale. Policlinico (sez. prat.) **34**: 1474, 1927.
11. Dünner, L., and Heilborn: Bilateral Phrenic Exeresis. Deutsche med. Wchnschr. **55**: 1918, 1928.
12. Iselin, M.: La Phrenicectomie bilaterale et les Problems Physiologiques qu'elle Pose. Medicine **12**: 389, 1931.
13. Anneuille, P.: Création d'un pneumothorax artificiel sur le coté de la paitrine opposé a une thoracoplastie. Bull. et mém. Soc. med. d. hôp. de Paris **51**: 337, 1927.
14. Maendl, H.: Phrenicoexairese und totale Brauersche Thorakoplastik links mit nachfolgenden pneumothorax rechts. Med. Klin. **25**: 1664, 1929.
15. Graham, E. A., and Bell, R. D.: Open Pneumothorax: Its Relation to the Treatment of Empyema. Am. J. M. Sc. **156**: 839, 1918.
16. Kinsella, T. J.: Bilateral Thoracoplasty, an Experimental Study Upon the Dog and the Monkey. Proc. Soc. Exper. Biol. & Med. **27**: 780, 1930.

THE RESULTS OF CAUTERY PNEUMOLYSIS OF ADHESIONS COMPLICATING ARTIFICIAL PNEUMOTHORAX*

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THE purpose of this very short paper on cautery pneumolysis is merely to summarize results and add some satisfactory figures to those already available in the hope of advancing further the use of this very valuable operation as an aid to artificial pneumothorax treatment.

Since the use of the Jacobaeus-Unverricht Thoracoscope and cautery has been so beautifully described by Matson in his elaborately illustrated article in the *American Review of Tuberculosis* of March, 1929, no time will be taken here to go into details of technic. The cases reported here have all been done by this method. The actual cautery has been used. It was intended some time ago to adopt the endothermic method but as results continued to be more and more satisfactory with the cautery as more skill was acquired in its manipulation, it is still being used. One of the most important factors in success with the use of the cautery is the use of very low heat, the tip never being heated even to a dull cherry red. This avoids too rapid cutting and the consequent danger of hemorrhage.

As more skill is acquired it is found that much larger and more extensive adhesions can be divided and complete collapse of the lung has been obtained in many cases which at first were considered unsuitable for treatment. It has also been found by experience that the x-ray, even when good stereoscopic films are available, is a very undependable guide as to whether or not adhesions can be cut. A considerable number of cases have been treated with complete success in which the x-ray films looked very discouraging. It has become our policy to look into the chest with the thoracoscope in all cases in which there seems to be any possibility of cutting adhesions, even when the odds seem strongly against success.

Gomenol was introduced into the chest following cauterization in about the first ten cases in the series. All of them, without exception developed fluid in considerable amount and no oil has been used in any of the later cases.

The figures are based on 141 cases operated upon since 1928. They have been done in four different hospitals: The General Hospital of Saranac Lake, N. Y., The Stonywold Sanatorium at Lake Kushaqua,

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N. Y., The U. S. Veterans' Hospital at Sunmount, N. Y., and The Metropolitan Life Insurance Company Sanatorium at Mount McGregor, N. Y.

The tabulated results are as follows:

A—Total cases attempted	141	
B—Number in which completely satisfactory collapse was obtained	122	or 87%
C—Serous fluid present before operation	30	or 21%
D—Serous fluid present after operation (including C)	64	or 45%
E—Number in which this was of serious consequence	9	or 6%
F—Number which developed purulent effusion without mixed infection	6	or 4%
G—Number which developed mixed infection—empyema (Both recovered)	2	or 1.5%

Before pneumolysis all of these cases of artificial pneumothorax were unsatisfactory due to the presence of the adhesions. In most of them the more actively diseased portion of the lung, usually that portion containing a cavity, in other words, the part of the lung most in need of compression, was the part held out by the adhesions. In 87 per cent of the cases complete collapse was obtained.

Complications: It will be seen that the development of serous effusion is the most common complication of the operation. The percentage of effusions however is no higher than the percentage in many series of pneumothorax cases in which pneumolysis is not done.

Of the number developing purulent effusion none came to opened drainage. Two developed acute staphylococcus empyemas but recovered completely after repeated aspirations, without drainage, and both are now well. In one case the lung was punctured but no fluid developed.

No case of hemorrhage occurred in the whole series. It is impossible to give the total number of adhesions cut but since the case with one single adhesion is unusual and the number found is apt to vary from two or three up to a dozen or more, it is fair to assume that in the series of 141 cases as many as five hundred individual adhesions have been cut. The danger from bleeding therefore is remote.

If this brief report of what seem to the author to be very satisfactory results will serve to encourage the use of cautery pneumolysis by any of those employing artificial pneumothorax, who have so far been unconvinced as to its soundness, or doubtful about its safety, the paper will have served its purpose.

THE SUSPENDED CAVITY IN ARTIFICIAL PNEUMOTHORAX*

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WHEN a lung containing tuberculous cavitation is being collapsed under artificial pneumothorax treatment, complete closure of these cavities is sometimes not possible until the maximum collapse of the lung has been obtained. In others this result is accomplished after only a small selective collapse has been produced.

In some patients the maximum collapse is desirable but is impossible of accomplishment because of several factors: thickness of cavity walls, consolidation of the surrounding lung tissue as in tuberculous pneumonia, or of adhesions between the lung and parietal pleura. If no factor other than adhesions prevents closure of a cavity, the adhesions are usually under tension and the cavity is suspended by it, creating what may be termed a hanging cavity. In these patients the lung tissue around the cavity has usually been very well collapsed, but the cavity with its string-like adhesions is plainly visible on the roentgenogram. Those cavities which are difficult of closure because of the thickness of their wall and surrounding tissue may have adhesions which are visible. This, however, does not mean that the cavities are held open by them, nor that closure would be more quickly obtained if these adhesions were severed. Thick wall cavities, also, often resist closure until the lung has been completely collapsed by means of positive pressure.

Many writers report that their best results and most permanent cures following artificial pneumothorax treatment are effected in those patients in whom a maximum collapse of the lung is obtained. They do not differentiate, however, between those patients in whom the maximum collapse is necessary to close cavities and those in whom the cavities are closed after a partial or selective collapse. It is not clear, either, from those reports whether those patients in whom only partial collapse was obtained were prevented from complete collapse by adhesions or other factors, or, if adhesions were the cause, whether these were such that might be severed or were too massive for successful treatment by operation. There are a considerable number of patients in whom the maximum collapse of the lung is neither necessary nor desirable, as the cavities will close, the sputum become negative, and the lesion heal with only a partial collapse. Any statistics, therefore, that attempt to show the results of treatment under the various degrees of collapse should take these factors into consideration.

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That it is desirable to obtain complete closure of all forms of tuberculous cavitation as soon and as safely as possible is, of course, obvious. As long as cavities remain open and the sputum positive there is constant danger of hemorrhage and bronchogenic spread, as well as the extension of disease to other organs. In hanging cavities there exists, also, the possibility that the tug of adhesions on the cavity wall will cause its rupture with the production of spontaneous pneumothorax, serous effusion, or empyema. During normal respiration in these patients the changing interthoracic pressures prevent incomplete rest to the lung. All of these dangers are increased if the diaphragm on that side is functioning normally, for on inspiration when the diaphragm descends it pulls the lung down with it and increases the tension on the adhesion. This is especially true, of course, if the base of the lung is attached to the diaphragm and the adhesions are at the apex. When there exists a combination of all factors—adhesions, consolidated lung and thick walled cavities—time only will give us an exact idea of the importance of the adhesions in the prevention of collapse.

There seems to be no unanimity of opinion as to just what course should be followed when adhesions exist. Some very excellent physicians believe that almost all adhesions should be severed if possible, while others believe that sufficient time should elapse to determine whether the adhesions will really defeat the purpose of the pneumothorax treatment. It does seem to me that insufficient data has been presented up to the present time on the ultimate fate of these patients under the various methods of procedure for one to state positively which is the best course to pursue; and, of course, it is only upon this ultimate fate that constructive conclusions can be drawn. It has been my experience that in the great majority of patients string-like adhesions can be stretched sufficiently by a continuation of the pneumothorax treatment alone to obtain the desired collapse. In many patients the addition of a phrenicectomy with the attendant rise in the diaphragm will cause enough relaxation of these adhesions to allow closure of cavities and sufficient rest to the lung. This is especially true if the adhesions are at the apex and base of the lung. By this procedure many hanging cavities are converted into closed cavities with only a relaxed adhesion left which is exerting no tugging on the cavity and which shows no tendency to keep it open. The attempt to overcome adhesions by these methods without opening the pleural cavity should be called the closed method. After sufficient time has elapsed and the closed method has not been effectual, we must decide between opening the thorax and severing the adhesions or doing a paravertebral thoracoplasty.

There are two distinct methods of open operation. One is by the Jacobeus operation in which the thorax is opened by inserting two trocars between the ribs into the pleural cavity. By this method ad-

hesions are located by the use of a thoracoscope inserted through one trocar while they are blindly cut by indirect vision with a cautery which is introduced into the pleural cavity through the other trocar. No attempt is made to ligate blood vessels that might be present. The other open method is one which gives direct vision of the pleural cavity and allows the operator to crush adhesions and use electrocoagulation or ligate them, if necessary, before they are severed. The technic of this operation as performed by me is as follows:

An incision about three inches long is made through the skin and fascia, usually over the second rib anteriorly. The pectoral muscle fibers

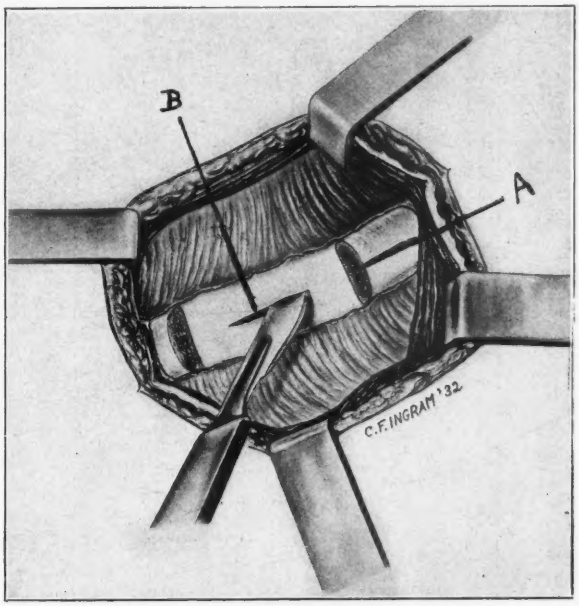

Fig. 1.—Incision through skin and fascia with muscles retracted. *A*, Cut end of rib removed subperiosteally; *B*, Incision (actual size) through periosteum and pleura into pleural cavity.

are separated and the rib exposed. Two or three centimeters of the rib are then removed subperiosteally. After complete hemostasis has been obtained, a small incision about three-quarters or one inch long is made through the periosteum and pleura into the pleural cavity. Small Cameron lights are then inserted through the opening which allows a perfect view of the interior of the thorax. The extent and number of adhesions can then be readily ascertained. If the Cameron lights are held behind the adhesion, one can usually see through them very clearly and determine if a pulmonary cavity extends out into the adhesions. Not infrequently blood vessels can also be readily identified. Two clamps are

inserted through the opening and the adhesions are crushed as far as possible from the lung tissue. These should be placed far enough apart to permit cauterization between them. If the adhesions are not too thick, they may be crushed for five minutes and then severed with the cautery knife between the clamps. Before the forceps are removed, electrocoagulation is used on the stumps. When this is done, the clamps are released

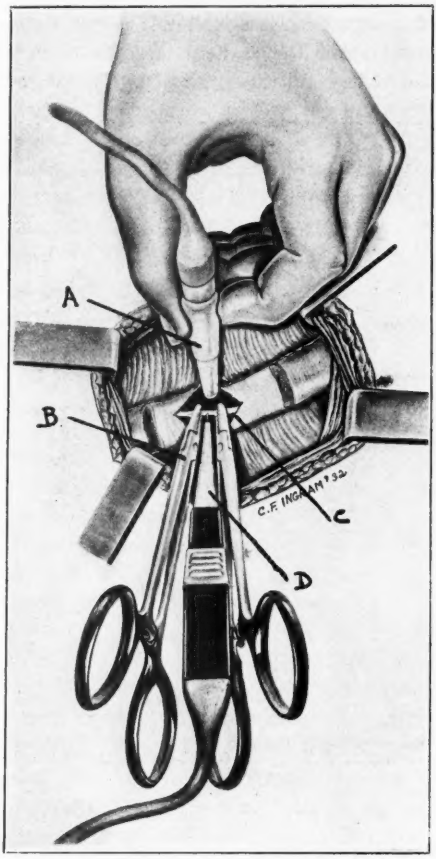

Fig. 2.—Pleural cavity opened. *A*, Cameron light held in pleural cavity; *B*, Hemostatic forceps shown crushing adhesion; *C*, Adhesion in pleural cavity; *D*, Cautery about to sever adhesion.

and the stumps examined for bleeding points. If the adhesions are very thick and it seems safer to ligate, then this can be done readily with the aid of a long tonsil needle. The sutures are to be placed in the crushed areas. After all adhesions have been severed the small opening in the pleural cavity is tightly closed with a fine needle and a No. 0 catgut to

prevent air leaks. The pectoral fibers are then approximated and the skin closed. I prefer this technic to the Jacobeus for several reasons. The first and probably the most important of these is that in my experience if sufficient time is allowed the closed method, and this should always be done, usually only those adhesions remain which cannot be safely cut without the precaution of crushing electrocoagulation or even

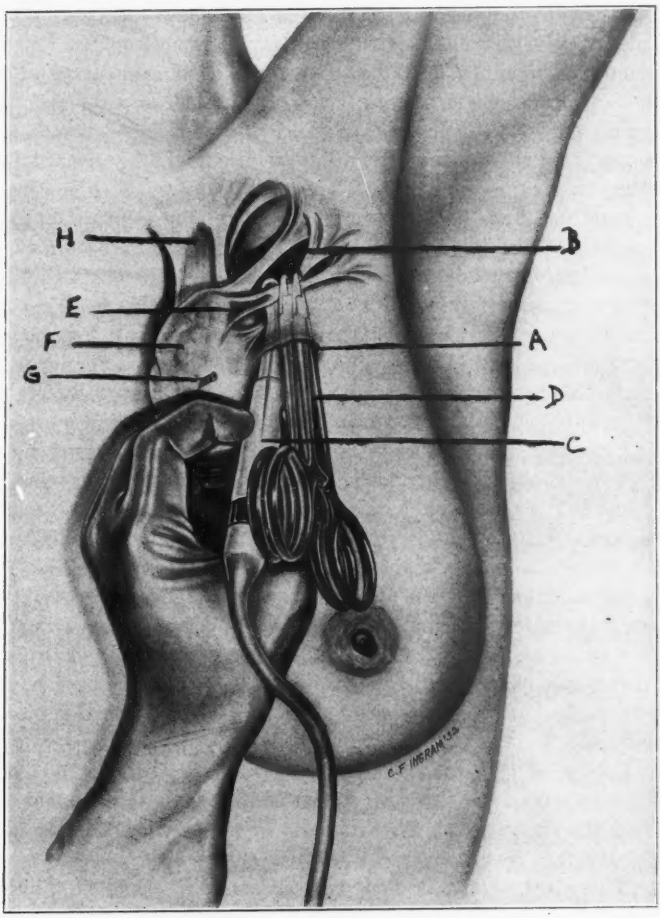

Fig. 3.—Composite picture showing incision, and view of the interior of pleural cavity as seen through the incision. *A*, Incision; *B*, Interior of pleural cavity; *C*, Cameron light (placed behind adhesion) showing extension of cavity into adhesion; *D*, Hemostatic forceps shown crushing adhesion distal to extension of cavity; *E*, Showing extension of cavity into adhesion as viewed with transillumination by means of Cameron light; *F*, Collapsed lung as seen through the incision; *G*, Stump of adhesion (severed in Fig. 2); *H*, Broad adhesion at apex with extension of cavity into it as viewed with transillumination. Note: All adhesions in this picture were severed.

ligature. Many smaller adhesions will have been stretched or relaxed sufficiently in most instances to allow the desired lung collapse.

Second in importance is the fact that the thoracic cavity can be readily explored by direct vision and the adhesions made more accessible. It is very rare indeed that one does not find the adhesions to be much thicker and broader than the roentgenogram has indicated, and with this incision one is prepared to handle the conditions as they present themselves. It seems to me, also, that there is less danger of infection of the pleural cavity with a clean-cut incision which is not much larger than the combined openings of the two trocars. One can carry on rigid technic, and there is less danger of cutting through an extension of the pulmonary cavity, the adhesion, or of leaving open blood vessels in the adhesions to be cut, or the intercostals which might be injured in the approach to the pleural cavity. This operation, as a rule, consumes much less time than the Jacobeus method, and the comfort of the patient must be considered. Bethune has modified the Jacobeus operation by using a very large thoracoscope which is inserted through a special trocar and applying clips to the adhesions before severing them. This seems to me much safer than the blind cutting by the usual Jacobeus method, and if these clips do not later act as a foreign body I should be inclined to use them as a modification of my technic. They could be applied through the small opening with more accuracy and ease. I am not, however, as yet convinced that these clamps may not later cause trouble. It is rarely necessary to make a very large incision or use rib spreaders to enlarge the opening as some operators describe, and I believe that most of the failures ascribed to this type of open operation are due to the difficulties in obtaining perfect closure of the pleural cavity. Pneumothorax cannot then be properly continued, and the lung reexpands and the space is lost. Two of my early failures were due to this difficulty of closure which resulted in leakage of air, emphysema, and finally opening of the wound. Since I have employed the technic just described, none of these difficulties has been encountered.

Dr. Carter, of Cincinnati, has described a very novel method of dissecting adhesions from their junction with the parietal pleura and then suturing the stumps. I think this would be an excellent idea if it were not for the fact that in most cases an unusually large incision is necessary. There are, no doubt, some cases in which it could be applicable. The small incision previously described overcomes the hazards of blind cutting of adhesions and usually allows sufficient room for careful work. I have had two experiences with hemorrhages, one of about two thousand cubic centimeters, after severing an adhesion without these precautions, so now I am very reluctant about cutting them blindly. If one believes, however, that it is safer to cauterize adhesions without first at-

fempting to prevent hemorrhage, then, of course, this may be accomplished also through this very small incision under direct vision, and if hemorrhage does occur one is in a position to control it. Excellent results by the open Jacobeus method have been reported by Jacobeus, Matson, Alexander, and many others, so that it seems that although the method described above is the procedure of choice, other methods give good results. The decision as to which adhesions should be cut, and when such cutting should be considered necessary, is also vitally important. There is a marked distinction between adhesions than can be cut and those which must be cut. It is of no particular interest that any given number of adhesions have been severed with ultimate closure of cavities with only a small mortality; if it is not clear that these adhesions were the cause of insufficient lung collapse, nor if such were the case, if the untoward effects of these same adhesions might have been overcome without surgical interference and no mortality had ensued.

In going over roentgenograms of some five hundred patients undergoing pneumothorax treatment I attempted to arrive at some conclusion as to what ultimately happened to patients with adhesions in whom no surgical procedure had been attempted. A survey of a large number of patients in whom these difficulties had been encountered, with a tabulation of their end-results, should be the foundation of any opinion, as it is what happens to the patient, after all, that is of the utmost importance.

In checking over this series of patients it was found that the number of adhesions which were seen depended on the length of time that had elapsed after the pneumothorax had been started. In a large percentage of the patients, early after the induction of the pneumothorax, some adhesions were present. It was also noted that if these same patients were viewed a few months later that most of the adhesions had either stretched or disappeared with nothing having been done except a continuation of the pneumothorax treatment. In another group it was noted that adhesions which were apparently holding out cavities were often sufficiently relaxed by the addition of a phrenicectomy to allow closure of the cavity and elimination of the sputum.

Out of 250 patients in whom artificial pneumothorax alone had been given sufficiently long to judge the end-results, it was found that after a few months of treatment only 20 remained in whom the adhesions were of such a nature as to suggest the necessity of intrapleural pneumolysis. Out of this group of 20 it was later found that in 11 of the patients the cavities had closed completely without pneumolysis in about eight months after the induction of the pneumothorax, and the sputum became negative and remained so after about six months. It also appeared that 2 others would close if positive pressures were used. Only 6 of the patients remained in whom adhesions were present that apparently needed to be severed. Three of these patients had already been referred for pneu-

molysis, but it was felt that the remaining 3 also should have been referred. In one patient the condition of the opposite lung seemed to make operative interference meddlesome.

It was apparent from this series that any decision as to the necessity of intrapleural pneumolysis would depend upon the length of time that they were viewed after the induction of the pneumothorax. It seems that the conservative method would be to wait until sufficient time has elapsed so that one can determine with more accuracy which adhesions can be cared for without surgery or whether other factors besides adhesions are the cause of failure to close the cavities. It also appears that in most cases from six to eight months of artificial pneumothorax treatment should be carried on before surgical interference is considered. The type of lesion and other factors should, of course, influence us in individual patients. In extensive soft lesions, especially in colored persons and others with little resistance, the maximum collapse of the lung as soon as possible is essential, and in this group it is probably justifiable to resort to pneumolysis earlier, especially if there exists a hanging cavity. It is generally believed that in such lesions there is a greater tendency for a spontaneous pneumothorax to occur while the adhesions are being stretched. In reality there seems to be less or at least no more danger of this happening while under pneumothorax treatment than in those patients not receiving it. In none of the patients reviewed had this occurred.

It is the contention of many, and there is some justification for this view, that as the purpose of the pneumothorax treatment is to cause closure of cavities as soon as possible all adhesions which are apparently preventing this result should be cut. This attitude, of course, could not be questioned if it were not for the fact that it is often possible to overcome the untoward effects of these adhesions more safely and in a short space of time without cutting them, and the added fact that many adhesions which are apparently preventing proper collapse of the lung are in reality not doing so. If a pneumolysis be performed on this group, and very little change is found in the cavity after operation, then the patient has been subjected to unnecessary surgical danger. In the series previously mentioned, no spreads were seen in the contralateral lung. There is probably less danger of this occurring from cavities in a partially collapsed lung than in one functioning normally. Undue haste, therefore, in cutting adhesions would seem to be unjustifiable.

In the clinics at Herman Kiefer Hospital, Maybury Sanatorium at Northville, and the American Legion Hospital at Battle Creek there are given over two thousand pneumothorax refills every month. Out of this large number of patients we have found it necessary over a period of five years to perform a pneumolysis to overcome adhesions in but 16 patients. It was because of the fact that in other clinics a much larger

number of patients had been operated upon from much less material that we undertook the survey mentioned to see if we had been too conservative. Our conclusions were that surgery should have been added earlier in but three of the patients as previously mentioned. In these patients the cavity was still open and the sputum positive after over a year of pneumothorax treatment. It is very difficult to determine exactly when the closed method may be considered a failure or that a cavity will not close unless adhesions are cut. We found one patient in whom the cavity was open at the end of fourteen months and apparently would continue so without surgical intervention, yet at the end of seventeen months the cavity had closed and remained so.

Of the 16 patients operated upon the cavities became closed and the sputum negative almost immediately in 11 patients. One has been operated upon too recently to report, but the cavities appear closed and it seems certain that an excellent result will be obtained. In another patient the lung was adherent at the base posteriorly over a considerable area, and while several adhesions holding out the apex were severed a complete collapse could not be obtained. The patient was considerably improved, however, and may yet obtain sufficient collapse. No complications arose in these patients aside from a small amount of serous fluid, which was not troublesome and usually very transient. In one patient this fluid was persistent but continued to be clear and uninfected for over two months. Constant aspirations were made, during one of which a needle was broken off into the pleural cavity and the cavity eventually became infected. In another patient with bilateral pneumothorax and fluid before operation the resident in an earnest attempt to prevent the space from being lost instituted positive pressures as high as +18; during a coughing spell the wound was broken open and leakage occurred with resulting empyema. In the two remaining patients, which were my first, the technic previously described was not used; instead, a wide incision was made between the ribs and complete closure of the pleural cavity was found impossible. Emphysema and leakage of fluid resulted, and the wounds finally broke down and became infected some time later. These patients died. All of them, however, were very poor risks, showing little or no resistance to their disease which was very extensive and soft. Tuberculous enteritis was also present, and the patients died months later of extension of their disease—plus this complication. The patients in whom only the closed method was used are in excellent condition with negative sputum.

The last word on this subject is, of course, not given here, but our experience with these patients is reported with the hope that it may lead to a more uniform selection of patients for intrapleural pneumolysis.

ACTINOMYCOSIS OF THE THORAX WITH REPORT OF A CASE SUCCESSFULLY OPERATED UPON*

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SUCCESSFULLY treated instances of pulmonary actinomycosis are so uncommon as to warrant a report of such an instance.

Mrs. E. B., age twenty-nine, University Hospital Number 57754, was admitted February 14, 1931. The patient gave a history of cough dating from November, 1929, followed by substernal pain. In March, 1930, she was delivered of a fullterm baby, and felt somewhat better for a short period following. Weakness, fever, sweats, anorexia, and loss of weight quickly supervened, however, and the patient gradually lost ground. In August, 1930, there was spitting of blood amounting to about an ounce and a half, and frequently since that time after coughing seizures, a slight amount of blood was expectorated. A small amount of yellowish sputum was raised from time to time. In October, 1930, pain and tenderness in the region of the third, fourth, and fifth ribs anteriorly on the left side was noted. In January, 1931, she entered the Sunnyrest Sanatorium for Tuberculosis at Crookston. Pneumothorax was attempted but it was not satisfactory. She was referred here by her physician, Dr. Paradis, with the impression of malignancy of the left lung and chest wall or obscure fungus infection. On admission here the chief complaints were a hacking cough with expectoration of a slight amount of mucopus, especially in the morning; dull steady aching pain in the left chest, breast and shoulder, with inability to raise the left arm because of pain, and weakness, fever, anorexia. The patient had lost thirty pounds in weight in the past year.

On physical examination the patient showed a well-developed physique, but was undernourished and appeared rather anemic. The slightest movement of the left shoulder girdle seemed to give her pain. Blood pressure was 120/60. The left chest was entirely dull with no breath sounds audible. The left breast was indurated and stood out rather prominently from the chest wall (Fig. 1) and was tender on the slightest touch. The abdomen was essentially negative as were the extremities and reflexes. The hemoglobin was 70 per cent; red blood cells 3,330,000; white blood cells 19,700; polymorphonuclears 82 per cent; lymphocytes 16 per cent, mononuclears 2 per cent. Blood urea nitrogen was 15. Blood Wassermann was negative. The urine was repeatedly examined and found to be consistently negative except for a few white blood cells. P.S.P. 65 per cent. Stool examinations were negative. Several sputum examinations showed the expectoration to be a thick whitish to yellowish sputum, occasionally tinged with blood, exhibiting clumps of pus on microscopic examination. Streptococci and staphylococci were found on smear and culture. No tubercle bacilli were found. The sputum was negative for actinomyces.

X-ray examination on February 16, 1931, showed evidence of a small hydropneumothorax in the left chest with evidence of displacement of the heart. There were some adhesions also at the left base. There was a mass in the left lung suggestive of lung tumor. (Fig. 2.) In a lateral film the mass appeared to extend to the anterior chest wall from the lung. Periostitis was noted along the superior margins

*From the Department of Surgery, University of Minnesota.
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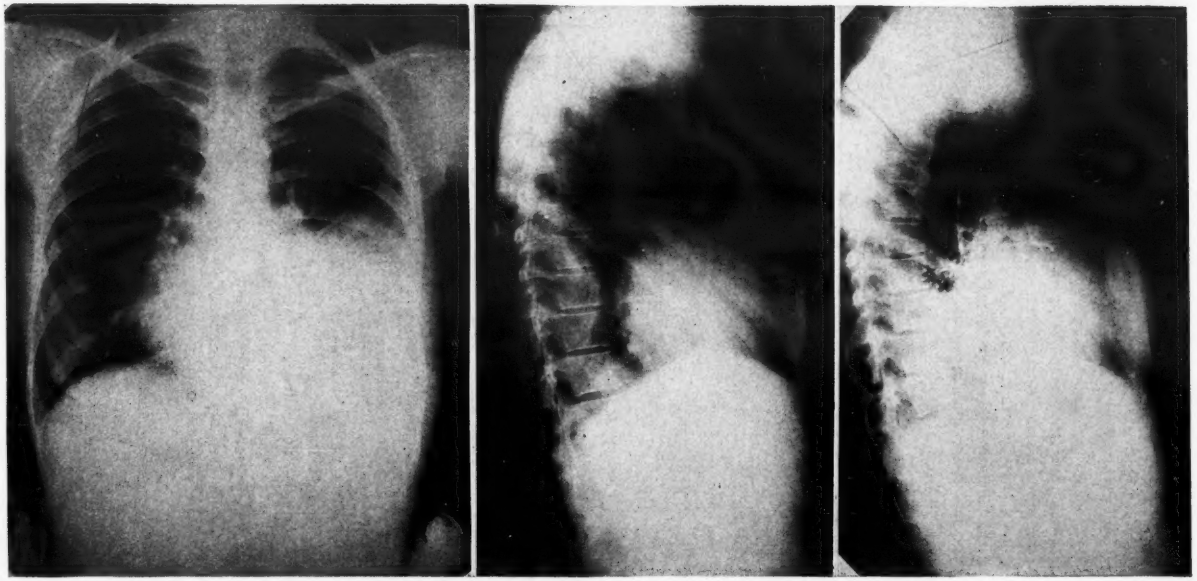
of the anterior portions of the sixth, seventh, eighth, and ninth ribs. A film was made on March 4, 1931, after the aspiration of iodized oil. The mass in the lower left lung was anterior to the left lower bronchus which was displaced somewhat backward and downward (Fig. 2 *C*). This bronchus was well filled out posteriorly and showed no intrinsic abnormalities. The upper lobe bronchus was also fairly well filled out and appeared to be displaced somewhat. Some compression of this bronchus was present. The appearance suggested that the anterior portion of the lower lobe bronchus was completely stenosed, and there was possibly some stenosis of the upper lobe bronchus also. None of the iodized oil entered into the region of the mass, indicating that it was a mass which had deeply penetrated into the chest cavity itself. Conclusion: Mass in the left lower lobe displacing bronchi. Probable stenosis of left lower lobe bronchus. Secondary periostitis of the sixth, seventh, eighth, and ninth left ribs anteriorly. Diagnosis: Probable malignancy of

Fig. 1.—Lateral photograph made just before operation. The small sinus in the chest wall was not present on admission, but represents the site of puncture and subsequent biopsy. Note how the breast stands up from the chest wall.

lung with invasion of chest wall. Stenosis of bronchi. Involvement of lung, chest wall, and ribs suggests actinomycosis (Dr. Leo Rigler). Roentgen examinations of the left shoulder revealed no evidence of a pathologic condition other than bone atrophy. The patient was examined with the bronchoscope on February 27, 1931, by Dr. K. Phelps. No abnormalities were seen in the larynx or trachea. The orifices of the right bronchi were not obstructed and no pus was seen; similarly no obstruction seemed to be present on the left, no stenoses of the bronchial orifices being observed.

I saw the patient on the Medical Service in consultation on March 5, 1931. The induration of the chest wall with periostitis of ribs, involvement of pleura and lungs suggested a diagnosis of actinomycosis. It was therefore believed best to insert a needle for aspiration. Previous aspirations had been made of the pleural cavity and a clear pleural transudate had once been obtained, which was negative on culture and smear for organisms. A large aspirating needle was thrust into the chest wall after anesthetizing the skin with procaine and a bloody fluid with sulphur-like granules was obtained. This was negative on direct examination for actino-

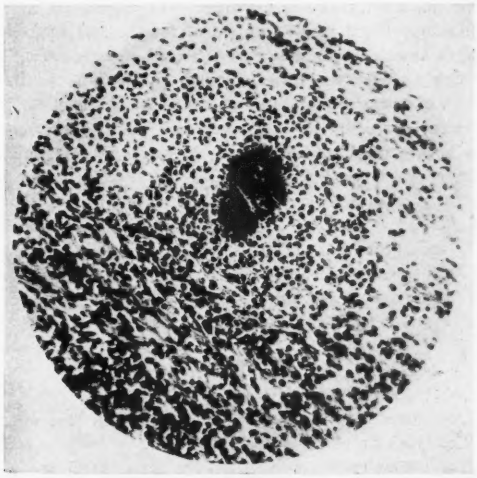


A *B* *C*

Fig. 2.—*A*, Roentgenogram of chest on admission (anteroposterior view). *B*, Lateral roentgenogram of chest on admission, showing large mass in left lung; periostitis of the anterior ends of the ribs below the breast shadow is apparent. *C*, The same after lipiodol filling. The lower lobe bronchus fails to fill anteriorly.

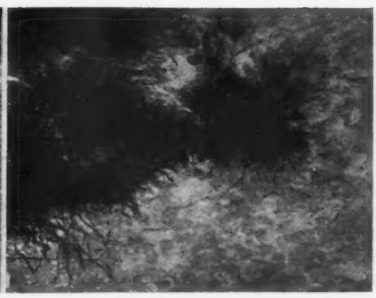
myces; however, after centrifuging and imbedding the sediment in paraffin several colonies were found on histologic examination of the stained sections (Fig. 3).

The patient ran a septic temperature vacillating between normal and subnormal readings to daily excursions usually exceeding 102° F. with a mean daily temperature running about 101°. The patient had complained of intermittent fever for more than



A

B



C

Fig. 3.—*A*, The ray fungus in a stained section (hematoxylin and eosin) obtained by centrifuging the aspirated exudate, imbedding the sediment in paraffin. The cellular zone of leucocytes about the colony and the more peripheral vascular and proliferative reactions are apparent. (×180.) *B*, A number of actinomyces colonies surrounded by leucocytes. *C*, A Gram's stain of a sulphur granule showing the intertwining mycelial threads of the streptothrix. (×400.)

a year before admission and had constant fever of the present grade for four months. The pulse was hurried and small and varied between 90 and 120 to 130, with a mean pulse rate somewhere between 100 and 110. A diagnosis was made of actinomycosis of the left lung with involvement of the pleura, ribs, chest wall, and left breast. The only procedure that seemed to hold out any promise of hope to the patient was successful extirpation of at least a part of the diseased tissue with effectual drainage.

However, the patient seemed a precarious risk for such a major undertaking. The condition was frankly discussed with the patient and she consented to operation. On March 11, 1931, the first operation was done.

Anesthesia, ethylene; preparation iodine and alcohol; incision semicircular just beneath the left breast. Operative procedure: Through this incision the breast was dissected upward, and a great quantity of pus escaped in which myriads of sulphur-like granules were apparent. The breast tissue was extensively undermined by this purulent accumulation. Segments of the third, fourth, and fifth ribs were excised from the cartilages anteriorly to well past the posterior axillary line. The pleura was unusually thick as were the ribs. A small portion of the thickened parietal pleura was excised, exposing the visceral pleura covering the lung. It seemed best to stop the operative procedure at this point, and a large gauze pack was placed into the wound to insure adequate hemostasis. It was my impression at the time that the breast would subsequently have to be sacrificed. It seemed best to proceed by graded operative procedure and not to do much at any one time, and to excise more tissue at a subsequent date. The patient left the table in good condition thirty minutes after the anesthesia was commenced.

Shortly after drainage of the chest wall the patient complained considerably less of tenderness in the left chest and fourteen days after operation slight amelioration of the temperature was noted. After eighteen days definite reduction in fever was noticed so that the daily excursions rarely went above 100° F. On April 6, 1931, the patient was transfused, 600 c.c. of unmodified blood being given.

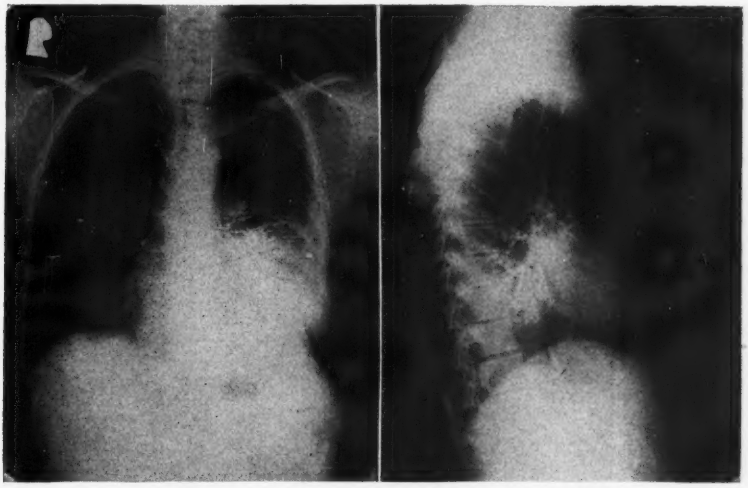
On April 17, 1931, the patient was reoperated upon. Anesthesia, ethylene; preparation iodine and alcohol; incision: the old incision was opened up together with a short vertical incision at the posterior end of the incision beneath the breast. Operative procedure: The breast was gradually freed from the pleura. A more liberal excision of the thickened parietal pleura beneath the area of previous rib resection was done. The visceral lamina of the pleura was then also incised and with surprising ease the greater portion of the upper part of the lung was completely decorticated, permitting it to expand immediately into the wound. No attempt was made to decorticate lower than the previous site of rib resection. The operation was interrupted at this point and the wound packed with gauze. There was rather free oozing due to the vascularity of the tissue. The skin was closed by a few interrupted sutures of linen. After this operation, there was slight increase of temperature again, the mean temperature running well above 100° with excursions going as high as 101°.

On May 7, 1931, the patient was operated upon again under ethylene anesthesia. The old wound was opened up and the sixth and seventh ribs were resected together with a good portion of the pleura lying beneath them. There was considerable oozing from the granulomatous tissue, which was satisfactorily staunched with hot moist packs and pressure. The ribs themselves were very much thickened and enlarged. Liberal sections of thickened parietal pleura were excised. An attempt was made to incise the visceral pleura and to decorticate the lower portion of the lung, but, owing to the presence of a parenchymatous lesion in the lower lobe, decortication was impossible. The heart within the pericardium was exposed during the procedure in the lower part of the wound. Adequate hemostasis was obtained, a small vaseline gauze pack was left in situ and the patient was transfused with 600 c.c. of unmodified blood on completion of the operative procedure.

Two days after this operative procedure there was a marked reduction in fever, the temperature rarely exceeding 99° F., with corresponding improvement in the patient's condition. The patient during this time expectorated some 25 to 100 c.c. a day; 25 c.c. being the usual amount charted. On June 3, 1931, a few small skin implants were implanted into the granulations overlying the pleura.

The patient was dismissed from the hospital June 24, 1931, very much improved. Prior to dismissal, it was believed wise to utilize all adjunct measures in the treatment and four deep x-ray treatments were given. One hundred and twenty-five per cent skin erythema dose was given over the anterior portion of the chest, the grafted area being covered. One hundred per cent skin erythema dose was given over the posterior and lateral portions, the treatments being given between June 10 and June 19, 1931.

The patient returned for observation at my request on November 6, 1931. The patient had been on massive doses of potassium iodide during her first hospital admission as well as during the summer after dismissal from the hospital. The patient felt much better during the summer and gained fifteen pounds in weight. There had been a continued discharge, however, from the small sinus at the posterior por-



A *B*

Fig. 4.—*A*, Röntgenogram of chest on most recent admission after lipiodol filling. There is still evidence of carnification in the lung. *B*, The same in the lateral view; contrast with Fig. 2 *C*. The anterior portion of the lower bronchus seen through the breast shadow does not fill as does the posterior division, but the bronchus itself is not obstructed.

tion of the wound. There was a crust at the upper part of the wound, while the remainder was well healed. The patient still complained of pain in the left chest, weakness, and of occasional palpitation. A hacking cough was still present, but there was less sputum. There was still slight tenderness in the left thoracic wall and the left chest was dull on percussion, and no breath sounds were heard in the lower portion of the chest. The hemoglobin was 63 per cent; red blood cells 3,920,000; white blood cells 13,050; polymorphonuclears 81 per cent; lymphocytes 17 per cent; mononuclears 1 per cent; eosinophiles 1 per cent. Urine was negative. Several pieces of tissue were curetted from the sinuses but failed to show actinomycosis histologically, only evidence of acute inflammation being present. X-ray examination revealed a very dense shadow over the whole left chest and evidence of previous rib excision. Lipiodol previously given was visualized in the chest. The patient was afebrile on this admission. Because of the complaint of pain in the lower chest and the persistent drainage from the sinus, it seemed best to open up the old wound.

This was done on November 11, 1931, under ethylene anesthesia. Methylene blue was injected into the sinuses on the chest wall, as we believed that any tract showing staining with methylene blue should be followed out. The upper wound failed to show any evidence of discoloration; the methylene blue penetrated deeply, however, into the chest beneath the thickened parietal pleura through the lower sinus. Segments of the eighth, ninth, tenth, and eleventh ribs were therefore excised together with the pleura over them. Considerable thick, purulent exudate fairly typical of actinomyces granules came out at the lower portion of the pleural cavity above the diaphragm. Generous segments of very thick parietal pleura were excised. At the upper portion of the wound where the breast had been reflected upward there appeared to be no definite lesion. The lower portion of the wound was packed with iodoform and plain gauze and the operative procedure terminated, fifty minutes

Fig. 5.—Photograph of patient on last admission. There is a small crust over the anterior portion of the original incision. The sinus posteriorly has not healed entirely, but there is no discharge.

after commencing the anesthesia. The patient left the table in good condition. This was the only operative procedure that lasted more than thirty minutes. The patient was transfused, 600 c.c. of unmodified blood being given after the operative procedure. After removal of the packs, the wound was freely irrigated daily with one-half strength tincture of iodine. On November 28, 1931, a few implant skin grafts were placed into the granulation tissue. The patient was dismissed on December 8, 1931.

The patient was admitted on March 4, 1932, without complaint for follow-up. The hemoglobin was 75 per cent; red blood cells 4,140,000; polymorphonuclears 67 per cent; lymphocytes 33 per cent. Urine was negative. X-ray examination still showed considerable density in the lower part of the left chest. (Fig. 4.) The patient stated that she had done the greater portion of her housework since her dismissal from the hospital and cared for four small children, the youngest of which is less than two years of age. She is free from fever, eats and sleeps well, has regained her initial weight, and has no complaints. There is no drainage from the wound.

(Fig. 5.) The patient was dismissed from the hospital with instructions to return in the fall for further observation.*

CASE SUMMARY

A woman of twenty-nine with symptoms of fifteen months' duration, with an extensive actinomycosis of the left thorax, involving lung, pleura, chest wall, and breast, has clinically recovered from the disease following four operative interventions for the removal of diseased tissue. Not enough time has yet elapsed to indicate whether she will remain permanently well from the ravages of the disease. The carnification in the lung will probably always persist.

MODES OF INVASION

Actinomycosis of the thorax constitutes about 15 per cent of all varieties of the disease observed clinically. The usual modes of involvement are: (1) aspiration of the invading organism through the bronchi into the lungs; (2) penetration of the esophagus (Soltmann, V. Baracz); (3) direct extension by contiguity, usually from the right abdomen (colon or liver); from a cervicofacial actinomycosis in the neck and rarely from actinomycosis of the breast or skin extending into the thorax, and, finally, (4) invasion of the lungs by generalization through the blood stream.

Of these modes of origin of thoracic actinomycosis, the first two may be said to be primary and the latter two are secondary. The manner in which the actinomyces obtain lodgement in the esophagus or lung is still a much debated issue. It is well known that streptothrix organisms resembling the pathogenic Actinomyces bovis may be natural inhabitants of the buccal cavity. Whether the disease arises as an autogenous infection from such organisms harbored within the mouth (Wright, Lord, Colebrook) or whether introduced from without, gaining entrance to the thorax by aspiration or swallowing is still a much disputed point even among those who have worked extensively in the field.

Davis, and Tunnicliff and Jackson appear to have demonstrated fairly conclusively that as far as the actinomyces-like organism in the tonsillar crypts is concerned, though there is a striking similarity to the Wolff-Israel organism responsible for actinomycosis in man, that the morphologic as well as cultural characters are distinctly different. The instances reported by Cope and the late McWilliams in which actinomycosis developed in wounds inflicted by teeth suggest rather strongly that organisms harbored in the buccal cavity of man may give rise to actinomycosis. In veterinary medicine it is well known that a large

*Since the above was written, the patient was readmitted (June 15, 1932) for the excision of a recurrence in the muscles of the back, just posterior to the site of the old incisions. There was no evidence of resistant actinomycosis in the lung. Actinomycosis, fortunately, exhibits an unusual tendency to burrow to the surface.

number of the cases of actinomycosis of cattle and swine are due to an actinobacillus described by Lignieres and Spitz (Griffith, Magnusson). Though the lesion produced by the actinobacillus cannot be distinguished from genuine actinomycosis due to the Actinomyces bovis, the response to treatment is entirely different, actinobacillosis reacting favorably to massive internal doses of iodine, whereas genuine actinomycosis of cattle remains refractory. The morphologic as well as cultural characters of the organism are, moreover, also distinctly different.

Henrici, who has written an authoritative text on "Molds, Yeasts and Actinomycetes," states that an unusually large number of varieties of actinomycetes of soil and animals have been described. He does not believe than an autogenous infection arising from actinomyces-like organisms in the mouth is responsible for actinomycosis of man.*

PATHOLOGY

Actinomycosis is a granulomatous infection exhibiting unusually aggressive invasive features, frequently destroying any tissue in its path. The lymph nodes seem to be spared† and are apparently only found involved in the generalized type (Werthemann). In actinomycosis generally, the site of invasion of the organism usually heals entirely, leaving little or no trace of the portal of entry. No matter which tissue is concerned, the reaction is fairly uniform and the pathologic effects are very similar. About the ray fungus, that obtains lodgement in tissue, is found a zone of cellular activity, in which are present large masses of polymorphonuclear and mononuclear leucocytes, epithelioid cells, and a rich network of young blood vessels. This reaction becomes manifest to the unaided eyes as abscess formation, with burrowing pus channels filled with purulent collections containing the yellow sulphur-granules (actinomyces colonies). Concurrently with this process of destruction, a tendency to healing goes on simultaneously, characterized by the proliferation of dense connective tissue more peripherally. The progression of the lesion by suppuration, degeneration, and liquefaction of tissue (abscess formation) and a peripheral tendency toward localization gives rise to a board-like induration in the affected tissues, with central areas of softening and consequent fistulous communications. The extension is usually so gradual that when a serous cavity, such as the pleura or peritoneal cavity, is invaded, a proliferative rather than an exudative reaction occurs. Effusions in serous cavities are therefore unusual in actinomycosis, though such instances are reported (see Table 2).

This tendency toward keloid-like formation of connective tissue is so

*Personal communication, April, 1932.

†In actinomycosis of cattle due to the actinobacillus the lymph nodes are constantly involved. Magnusson states that this occurrence alone serves to differentiate the lesion from genuine actinomycosis.

striking in actinomycosis that not uncommonly the lesion, whether in the neck, chest, or abdomen, is mistaken on its clinical features for malignancy. The vascularity of the granulomatous reaction of actinomycosis at operation is always striking.

Actinomycosis of the lung and pleura is essentially a destructive lesion, in which the early manifestations are not unlike those of a bronchopneumonia with abscess formation. Canali has reported an instance of pulmonary actinomycosis in which only a bronchitis with purulent sputum occurred. Skworzoff's case of so-called bronchitic actinomycosis was found on postmortem examination to have bronchiectatic cavities in the lungs. Inasmuch as Thompson found a streptothrix in the sputum of 65 per cent of 1000 patients with asthma, bronchitis, bronchiectasis, and tuberculosis, unless the organism is culturally and morphologically identified as the Israel organism of actinomycosis, its presence cannot be looked upon as synonymous with pulmonary actinomycosis.

Kirklin and Hefke from a roentgenologic standpoint have classified pulmonary actinomycosis according to its extent, which would also serve as a satisfactory basis for a pathologic grouping. Four types may be recognized; viz., pulmonary, pleural, pleuropulmonary, and thoracic. Extension into the pericardium and even the heart (Letulle and Hufnagel, Kasper and Pinner) may occur. Extensions through the diaphragm are not uncommon, and erosion of the sternum ribs and vertebral bodies are not infrequently seen. Invasion of the spinal canal with paraplegia has been observed. The lower lobe of the lung is the most frequently affected, but the apices of the lung do not escape. Whereas West found the left side the most frequent site of the disease, Good found pulmonary actinomycosis to be equally frequent on the right.

In their 14 instances of pulmonary actinomycosis, Kirklin and Hefke found evidence of an antecedent abdominal actinomycosis in 8 cases, from which source the pulmonary lesion was believed to have developed by direct extension. In one instance, the lesion extended to the thorax from the neck. Wildenburg has reported a similar instance, and another has been observed at the University Hospital here when the conservative treatment of actinomycosis of the neck with potassium iodide and x-rays was practiced.

Inasmuch as actinomycosis generalizes largely by the blood stream, the lungs are usually early involved when a distant spread of the disease occurs from other parts. Late in the course of thoracic actinomycosis, a similar generalization from the lungs to distant parts by way of the blood stream may occur.

SYMPTOMS

Primary actinomycosis of the lungs usually mimics the more common forms of pulmonary suppuration in its manifestations. The onset may

be rather abrupt, suggesting pulmonary abscess, but more frequently insidious and gradual in its development as observed in bronchiectasis or tuberculosis.

Fever, cough, and expectoration are constant accompaniments of actinomycosis of the lung. In those instances where the esophagus was the primary site of lodgement of the organism, a history of dysphagia may precede the onset of pulmonary suppuration. Fever in actinomycosis is dependent in large measure on the degree of secondary infection with pyogenic invaders, and varies also with the extent of thoracic involvement. Cough may be slight or harassing. It is usually accompanied by expectoration, but is occasionally nonproductive. Marked hemoptysis rarely occurs though blood-streaked sputum is not uncommon in primary pulmonary actinomycosis. The sputum as in all types of pulmonary suppuration is thick and viscid, and occasionally the sulphur-like granule of the ray fungus may be identified in the expectoration.

As soon as the pleura is involved, intense constant pain is frequently complained of, and the patient avoids any exertion that demands extra excursions of the thoracic muscles of respiration. Dyspnea is not infrequent. When penetration of the endothoracic fascia into the chest wall occurs, tenderness is usually manifest. Lord states that fistulous openings in the chest wall are present in about 82 per cent of cases.

Sweats, anorexia, loss of weight and strength are fairly constant sequels of the pulmonary suppuration, and are always in evidence in extensive or late cases. Weakness is often a dominant complaint.

The following analysis of symptoms in 40 cases of primary actinomycosis of the thorax recorded in the literature was made by Good:

TABLE I

SYMPTOMS	PRESENT	NOT RECORDED	ABSENT	DOUBTFUL
Fever	34	1	1	4
Cough	36	1	—	3
Pain	38	2	—	—
Expectoration	27	4	4	5
Hemoptysis*	3	5	17	5
External abscess	32	2	6	—
Sinuses	23	8	9	—
Loss of weight†	30	5	2	2
Involvement of ribs	10	10	9	11
Involvement of vertebrae	4	—	—	—
Involvement of sternum	1	—	—	—
Anemia	20	6	2	10

*In 10 cases the sputum was streaked with blood.
†One patient gained weight.

DIAGNOSIS

Without direct isolation of the organism from the sputum or by aspiration through the chest wall with a needle, the diagnosis will always re-

main in doubt. The roentgen ray evidence of periostitis or local areas of destruction of ribs, vertebrae, or sternum in the presence of a lesion in the lung with pleural involvement is highly indicative of pulmonary actinomycosis. It is usually only in the presence of external fistulous communication that this finding is present. Only 5 of 14 cases of pulmonary actinomycosis reported by Kirklin and Hefke, however, exhibited this finding. Though abscess formation is constant in pulmonary actinomycosis, they are rarely large enough to be detected with the roentgen ray. And the intense carnification of the lung about the small cavities precludes their being easily demonstrated.

The diagnosis of actinomycosis of the lung is rarely made early, and is usually deferred until the organisms are found or the roentgen criterion just described obtains. Frequently when the organisms cannot be identified by microscopic examination of the fresh exudate, they may be found with ease by imbedding the sediment obtained after centrifuging in paraffin and staining a cut section with hematoxylin and eosin (Fig. 3).

Just as the biologic test for tuberculosis by guinea pig inoculation is a more sensitive test than the isolation of the tubercle bacillus from urine, sputum, or other exudates, similarly the stained tissue section method in most hands presents distinct advantages over the microscopic examination of fresh exudate when sulphur granules are not obviously present. The superiority of the tissue method is in part due to the fact that a more systematic search is made for the colonies in a stained section. If a sinus is present, its walls may be curetted or a piece of tissue may be excised for biopsy, either of which usually affords reliable evidence of the presence or absence of actinomycosis.

When sinuses have long been present in actinomycosis, the organism may be very difficult to find, and the one who taps the lesion first with a hypodermic needle has the best opportunity to isolate the organism in the fresh preparation.

Attempts have been made by Colebrook and others to develop a diagnostic serologic test for actinomycosis, a method that as yet, however, has not come into general use.

DIFFERENTIAL DIAGNOSIS

Pulmonary abscess, bronchiectasis with abscess or carnification of the lung, empyema, tuberculosis, and malignancy of the lung and pleura are the most common lesions from which actinomycosis has to be differentiated. Unresolved pneumonia, syphilis of the lung, blastomycosis, and hydatid cyst of the lung may rarely give rise to confusion. Early actinomycosis of the lung in the absence of isolation of the organism cannot be differentiated with certainty from any of these lesions. Careful inspection of stereoscopic films of the chest and deliberate search

for the actinomycees are the most helpful agents in the differential. In well-established cases of actinomycosis of the thorax, there should be little cause for confusion. It is surprising how often, however, cases of actinomycosis of the head and neck are referred to the hospital with the diagnosis of malignancy, either carcinoma or sarcoma.

PROGNOSIS

Thoracic actinomycosis has the worst outlook of any form of the localized forms of actinomycosis. In a report of 87 cases of actinomycosis seen in Norway, Harbitz and Grondahl stated that the thorax was involved in 23 per cent; all of these patients died. In his article, Good states: "The disease is almost always eventually fatal." He reported 13 cases from the Mayo Clinic of which 6 or 47 per cent were dead at the time of the report. All except 3 were followed. In one case there was striking improvement, and the disease appeared to have become arrested. Very few clinics that have reported their results in the treatment of actinomycosis have any successes in the thoracic group.

Generalization is not infrequent in untreated thoracic actinomycosis. Not infrequently, however, death results from the pulmonary suppuration and effects incidental to the debility of the disease. Actinomycosis of the thorax occasionally runs an acute course. In his study, Good found that in 27 cases reported in the literature in which information was obtainable on this point, that the average duration of symptoms from onset to death was eight months. In 7 of 13 cases observed at the Mayo Clinic of which number 6 were dead when Good made his report, the length of life after onset of symptoms was twenty-nine months. Vinson and Sutherland report the instance of a woman aged thirty-seven years with an esophagobronchial fistula due to actinomycosis in which the initial lesion had apparently started in the right tibia twenty-eight years before.

TREATMENT

The therapeutic agents that have proved of most value in the treatment of actinomycosis are surgery, potassium iodide, and the roentgen rays and radium. A few have reported surprisingly good results with an autogenous vaccine prepared from the exudate (Wynn, Collie, Malcolm).

The internal administration of potassium iodide in veterinary medicine in the treatment of bovine and swine actinomycosis led van Itterson in 1892 to adapt its use to human forms of actinomycosis.

As pointed out above, however, it is only in the actinobacillosis of cattle and swine that the internal administration of massive doses of iodine is followed by dramatic effects (Magnusson). In the actinomycosis of cattle, however, in which the Israel type of organism is the causative agent, iodides do not possess the virtues of a specific. Dr. Fitch, profes-

sor of veterinary medicine in the College of Agriculture at the University of Minnesota informs me that since this distinction has been made in the common forms of bovine actinomycosis potassium iodide has lost a great deal of its prestige in the treatment of genuine actinomycosis. As a single therapeutic agent in human actinomycosis potassium iodide is certainly of doubtful value.*

The use of x-ray treatment in the management of actinomycosis was first made by Harsha of Chicago in 1904, and later by Bevan. Until 1913 when Levy of Küttner's Clinic in Breslau revived its use as a therapeutic agent, little credence was given to x-rays as a therapeutic measure of value in the treatment of actinomycosis. In more recent years, irradiation as a therapeutic agent has been extolled by Heyerdahl, Jüngling, New and Figi, and Desjardins. In most of the cases that they report, however, irradiation has been used in combination with surgery and the internal administration of potassium iodide. New and Figi particularly advise massive internal doses of iodides to be given simultaneously, and emphasize the necessity of surgical incision, curettement, drainage, and excision of involved tissue. Heyerdahl, Jüngling, and Desjardins, however, seem quite willing to dispense with all forms of treatment except irradiation.

The only well-authenticated case that I can find in the literature in which an instance of pulmonary actinomycosis was cured with the employment of the x-rays as the principal therapeutic agent is that of Marko, listed in the table of the reported cured cases. A large number of reports are available, however, indicating the futility of roentgen treatment in pulmonary actinomycosis.

Forty years ago, Schlange, then an assistant in Von Bergmann's clinic in Berlin, pointed out that now and then patients with actinomycosis exhibit an unusual tendency to get well spontaneously when aided by surgical drainage. Complete surgical excision in the manner in which one deals with malignancies is not necessary, but the promotion of surgical drainage combined with liberal excision of diseased tissue usually results in cure. Prior to 1929, actinomycosis at the University Hospital was dealt with largely by x-ray treatment, internal administration of massive doses of potassium iodide, and surgical restraint. Incisions were made as they seemed indicated. Since then 5 cases of actinomycosis have been observed, 4 of the cervicofacial type, and the thoracic case here reported. All have been dealt with surgically, and all are well. The only patient that received any x-ray treatment was the thoracic case, it being considered that all adjuvant measures should be employed in so desperate a case.

One cannot escape the impression as he peruses the literature of

*Dr. A. T. Henrici informs me that actinomycosis due to the actinobacillus has never been observed in human beings, though Colebrook states that Pinoy observed a case in France. Thompson and Willius recently report another (J. A. M. A. **99**: 298, 1932.)

TABLE II

COLLECTED CASES OF ACTINOMYCOSIS OF THE LUNG REPORTED CURED IN THE LITERATURE

AUTHOR	YEAR	DESCRIPTION OF LESION	TREATMENT	RESULT
Schlange	1892	Male age fifty-four; extensive actinomycosis of right lower chest	v. Bergmann made extensive incisions; 2 ribs resected	Well six years later
Jakowski	1897	Female age twenty-nine; right-sided pleuropulmonary lesion	Resection of sixth to ninth ribs (Jawdynski)	Well; report only twenty-three days after dismissal
Horn (quoted by Karewski 1914)		Details missing	Operation by Perthes	Well one year later
Karewski	1898	Adult male; extensive actinomycosis of right thorax	Extensive removal of skin, fascia, and muscle of chest wall with rib resection	Well sixteen years later
Brentano	1905	Male age twenty-four; left-sided lesion	Resection of eighth, ninth and tenth ribs; cauterization	Well one year later
Hamm (referred to by Shiota, 1909, and Karewski, 1914)	1906	Pulmonary actinomycosis; treated as empyema	Operated upon by Ledderhose	Well one year later
Trevithick	1906	Boy age sixteen; pulmonary actinomycosis left lung; later abdominal actinomycosis	Incision, x-ray, KI (Author attributes improvement to spontaneous cure)	Well one year later
Wynn	1908	Boy age fourteen; actinomycosis of left chest; later abdominal actinomycosis	Rib resection; autogenous vaccine	Well one year later
Shiota	1909	Male age thirty-two; actinomycosis of both sides of chest with interval between	Operated upon both sides of chest; several rib resections	Well ten months later
Opokin	1909	Case of Rasumousky (1899) details lacking	Surgery	Recovery
Lexer	1910	Right lung and pleura	Extensive excision; six ribs resected; cauterization	Recovery

TABLE II. (Cont'd)

AUTHOR	YEAR	DESCRIPTION OF LESION	TREATMENT	RESULT
Bauer	1913	Boy age six; lower left lung lesion later abdominal actinomycosis	Incisions; curettement, drainage by Lexer	Recovery
Collie	1913	Male age forty; actinomycosis left groin and left chest wall	Incisions and treatment by vaccines (Sir Almroth Wright)	Well six months later
Sauerbruch	1920	Actinomycosis of lung; details missing	Surgery	Well four years later
Bigland and Sergeant	1923	Male age twenty-two; developed actinomycosis of pericardium and left pleural cavity after pneumonia	Drainage and irrigations with iodine	Recovery
Lord	1926	Male age 39; actinomycosis of left lung with organism in sputum	Drainage of pulmonary abscess	Well four years later
Schweizer	1929	Male age thirty-three; diagnosed tuberculosis of left lung for two years	Two operations; extensive rib resection	Well
Marko	1929	Adult male; antecedent cervical actinomycosis; treated with surgery, KI, and x-ray successfully; four months later developed left lung lesion with actinomycosis in sputum	X-ray and KI	Well two years later
Wangensteen	1932	Female age twenty-nine; ill fifteen months, extensive lesion of left lung; pleura, chest wall, and breast	Four operations; extensive rib resection and excision of parietal pleura; KI; one course x-ray treatments	Well six months later
COLLECTED CASES OF ACTINOMYCOSIS OF THE LUNG REPORTED DEFINITELY IMPROVED IN THE LITERATURE				
Torek	1926	Male age forty-two; pain in left chest for one year; extensive lesion; previous hemoptysis	Several operative procedures; KI	Remarkably improved
Good	1929	Details missing		Arrested

TABLE II. (Cont'd)

AUTHOR	YEAR	DESCRIPTION OF LESION	TREATMENT	RESULT
*Recovered Instances in Which Actinomyces Were Found in Pleural Effusion**				
Schlange	1892	Actinomyces in sputum and pleural exudate; previous parametritis	Single aspiration	Recovery
Nakamura (quoted by Shiota)	1909	Male age fifty-three; pleural exudate in which actinomyces were found	Aspiration	Well five months later
Geymüller	1929	Male age forty-five; acutely ill; aspiration of right subphrenic and pleural space; actinomyces found also in serosanguinous exudate	Two aspirations and KI	Well two and one-half years later
Instances Reported Cured in Which Details Are Lacking				
Bichowski (quoted by Opokin, 1909)	1896	No details—not listed by Karewski (1914)		
Krause		Adult male; actinomyces in sputum; Diag. pleuropulmonary actinomycosis; no other data	KI and x-ray	Reported well; apparently recent case

*In these instances the only apparent evidence of actinomycosis was the isolation of the actinomyces in a serosanguineous pleural exudate. Actinomycosis so regularly gives rise to a productive reaction that these cases are to be looked upon as unusual; ascites in abdominal actinomycosis is a rarity.

actinomycosis that favorable results in thoracic and abdominal groups are less often reported since x-rays have been more relied upon in the treatment. It is not to be denied that irradiation is of value in actinomycosis. The report of Heyerdahl with reference to the cervicofacial group establishes its worth beyond question. It is to be emphasized, however, that though irradiation is of value in the treatment of this granulomatous condition, that the mainstay of treatment is surgery. When less time is lost in dilatory methods of dealing with thoracic and abdominal actinomycosis, and earlier recourse is had to surgical measures, cures will be far more numerous.

The reported cures in thoracic actinomycosis are indicated in the accompanying table. (Table 2.) Undoubtedly this list is incomplete, but it indicates nevertheless how rarely actinomycosis of the thorax has been dealt with successfully.

Surgery is not to be recommended in thoracic actinomycosis with evidence of generalization. No definite operative plan can be elaborated in attacking thoracic actinomycosis surgically. On the whole, the principles outlined above; viz., surgical drainage together with sacrifice of diseased tissue usually suffices. The improvement attendant upon the graded operation will determine how much should be done.

SUMMARY AND CONCLUSIONS

An instance of extensive primary thoracic actinomycosis with involvement of lung, pleura, chest wall, and breast, is reported in which cure resulted following radical surgery. Four operative interventions with liberal excision of diseased tissue were done. The other instances of reported cures in thoracic actinomycosis are collected.

It is pointed out that surgical measures still constitute the chief therapeutic measure of relief in dealing with actinomycosis. It is believed that too much reliance upon the adjuvant measures of irradiation and internal administration of potassium iodide has contributed to poor results in abdominal and thoracic actinomycosis. Cures will be more frequently reported when consistent and early recourse is had to surgery in thoracic actinomycosis.

REFERENCES

1. v. Baracz: Ueber die Aktinomykose des Menschen auf Grund eigener Beobachtungen, Arch. f. klin. Chir. **68**: 1050, 1902.
2. Bauer: Chirurgische Behandlung der Lungenaktinomykose, Mitt. a. d. Grenzgeb. d. Med. u. Chir. **25**: 135, 1913.
3. Bevan, A. D.: Actinomycosis, Ann. Surg. **41**: 641, 1905.
4. Bigland, D., and Sergeant, F.: A Case of Actinomycosis With Recovery, Brit. M. J. **2**: 61, 1923.
5. Bostroem: Untersuchungen ueber die Aktinomykose des Menschen, Ziegler's Beiträge **9**: 1, 1891.
6. Brentano: Lungenaktinomykose, Deutsche med. Wchnschr. **32**: 165, 1906.
7. Canali: Quoted by Hektoen.
8. Colebrook, L.: The Mycelial and Other Microorganisms Associated With Human Actinomycosis, Brit. J. Exper. Path. **1**: 197, 1920.

9. Collie, J.: A Case of Actinomycosis Treated by a Vaccine, Brit. M. J. **1**: 991, 1913.
10. Cope, V. Z.: A Clinical Study of Actinomycosis With Illustrative Cases, Brit. J. Surg. **3**: 55, 1915.
11. Davis, D. J.: The Actinomyces-Like Granules in Tonsils, J. Infect. Dis. **14**: 144, 1914.
12. Desjardins: Radiotherapy in Actinomycosis, Radiology **11**: 321, 1928.
13. Fitch, C. P.: Personal communication. April, 1932.
14. Geymüller: Zur Kasustik der Aktinomykose innerer Organe, Schweiz. med. Wchnschr. **58**: 635, 1928.
15. Good, L. P.: Actinomycosis of the Thorax, Ann. Surg. **21**: 786, 1930.
16. Griffith, F.: On the Pathology of Bovine Actinomycosis, J. Hyg. **15**: 195, 1916.
17. Hamm, A.: Diagnostik der Aktinomykose, Deutsche med. Wchnschr. **32**: 947, 1906.
18. Harbitz, F., and Grondahl, N. B.: Actinomycosis in Norway; Studies in the Etiology, Modes of Infection and Treatment, Am. J. M. Sc. **142**: 386, 1911.
19. Harsha, W. M.: Actinomycosis of the Jaw, Ann. Surg. **39**: 454, 1904.
20. Hektoen, L.: Actinomycosis of the Respiratory Tract, Internat. Clin. **2**: 97, 1901.
21. Henrici, A. T.: Molds, Yeasts, and Actinomycetes, New York, 1930, John Wiley & Sons.
22. Heyerdahl, S. A.: Actinomycosis Treated With Radium, J. A. M. A. **73**: 1928, 1919.
23. ——: Actinomycosis of the Face and Neck Treated With Radium, Brit. J. Radiol. **31**: 1, 1926.
24. Hodenpyl, Eugene: Actinomycosis of the Lung (J. M. Smith Prize Essay in 1890), Med. Record. **38**: 653, 1890.
25. Israel, Jones: Klinische Beiträge zur Kenntnis der Aktinomykose des Menschen, Berlin, 1885, August Hirschwald.
26. Jakowski, M.: Aktinomykose der Lunge und Pleura, Centralbl. f. Chir. **24**: 480, 1897.
27. Jüngling, V.: Zur Röntgenbehandlung der Aktinomykose, Brun's Beiträge **118**: 105, 1920.
28. Karewski, F.: Beitrag zur Lehre von der Aktinomykose der Lunge und des Thorax, Berl. klin. Wchnschr. **35**: 328, 350, 372, 1898.
29. ——: Discussion of Brentano's Paper, Deutsche med. Wchnschr. **32**: 165, 1906.
30. ——: Die Chirurgische Behandlung der Lungen aktinomykose, Verhandl. d. deutsch. Gesellsch. f. Chir. **37**: 541, 1907.
31. ——: Die Aktinomykose der Lunge und der Pleura, Ergebn. d. Chir. u. Orthop. **8**: 424, 1914.
32. Kasper, J. A., and Pinner, Max: Actinomycosis of the Heart; Report of a Case With Actinomycotic Emboli, J. A. M. A. **10**: 678, 1930.
33. Krause, P.: Zur Röntgen diagnostik und Röntgentherapie der Lungenaktinomykose, Fortschr. a. d. geb. d. Röntgenstrahlen. **40**: pt. 2, 1114, 1929.
34. Kirklin and Hefke: The Roentgenologic Aspects of Actinomycosis of the Lungs, Am. J. Surg. **13**: 1 (July), 1931.
35. Letulle, M., and Hufnagel: L'Actinomycose du coeur, Bull. Acad. de méd. Paris **82**: 120, 1919.
36. Levy: Röntgenbestrahlung der Aktinomykose, Centralbl. f. Chir. No. 4, p. 121, 1913.
37. Lexer, E.: Demonstration in der chirurgische Universitätsklinik, Centralbl. f. Chir. No. 47, p. 1499, 1910.
38. Lord, F. T.: A Contribution to the Etiology of Actinomycosis: The Production of Actinomycosis in Guinea Pigs Inoculated With the Contents of Carious Teeth, Bost. M. & S. J. **163**: 82, 1910.
39. ——: The Etiology of Actinomycosis: The Presence of Actinomycetes in the Contents of Carious Teeth and the Tonsillar Crypts of Patients With Actinomycosis, J. A. M. A. **55**: 1261, 1915.
40. ——: Discussion of the Diagnosis in a Case of Primary Pulmonary Actinomycosis, M. Clin. North America **8**: 1485, 1924.
41. ——: Discussion of Torek's Paper, Arch. Surg. **12**: 436, 1926.
42. Malcolm, W. P.: Vaccine in Mediastinal Actinomycosis, Brit. M. J. **2**: 488, 1916.
43. Magnusson, H.: The Commonest Forms of Actinomycosis in Domestic Animals and Their Etiology, Acta. path. et microbiol. Scandinav. **5**: 170, 1928.

44. Marko, D.: Beiträge zur Röntgendiagnostik und Therapie der Lungenaktinomykose, Fortschr. a. d. Geb. d. Röntgenstrahlen. **39**: 629, 1929.
45. McWilliams, Clarence: Actinomycosis of Phalanx of Finger, Ann. Surg. **66**: 117, 1917.
46. Naussac, J.: The Pathology, Symptomatology, and Differential Diagnosis of Pulmonary Actinomycosis, Internat. Clin. **3**: 1, 1921.
47. New, G. B., and Figi, F. A.: Actinomycosis of the Head and Neck, Surg. Gynec. & Obst. **37**: 617, 1923.
48. Oldenburg, F.: Beitrag zur Klinik der primären Lungen Aktinomykose, Beitr. z. Klin. d. Tuberk. **74**: 92, 1930.
49. Opokin, A. A.: Lungen und Pleuraaktinomykose nach der Beobachtungen Russischer Autoren, Arch. f. klin. Chir. **88**: 460, 1909.
50. Sauerbruch, F.: Die Aktinomykose der Lungen. Die Chirurgie der Brustorgane, Berlin, 1920, J. Springer, p. 838.
51. Schweizer, R.: Demonstration eines geheilten Falles von Aktinomykose der Lunge, Pleura, und Brustwand, Schweiz. med. Wchnschr. **59**: 249, 1929.
52. Shiota, H.: Beitrag zur Kenntnis der Menschlichen Aktinomykose, Deutsche Ztschr. f. Chir. **101**: 289, 1909.
53. Skworzoff, M. A.: Ueber die sogenannte "oberflächliche" form der primaren Lungenaktinomykose, Virchow's Arch. **261**: 503, 1926.
54. Soltmann: Ueber Aetiologie und Ausbreitungs bezirk der Aktinomykose, Jahrb. f. Kinderh. **24**: 129, 1886.
55. Thompson, E. T.: Note on an Anaerobic Streptothrix Occurring in Sputum in Pulmonary Diseases, Lancet **2**: 1036, 1929.
56. Torek, F.: Actinomycosis of the Lungs and Chest, Arch. Surg. **12**: 385, 1926.
57. Trevithick: A Case of Actinomycosis. Primary Abscess of Left Lung Exhibiting Cure; Metastatic Manifestation in the Pelvis Declaring Itself After a Year's Latency With Abscess Formation and Slow Spontaneous Cure, Lancet **2**: 158, 1906.
58. Tunnicliff, Ruth, and Jackson, Lelia: Vibriothrix tonsillaris. N. S. P. The Organism of Actinomyces-like Tonsillar Granules, J. Infect. Dis. **46**: 2, 1930.
59. Vinson, P. P., and Sutherland, C. G.: Esophagobronchial Fistula Resulting From Actinomycosis; Report of a Case, Radiology **6**: 63, 1926.
60. Werthemann, A.: Ueber die Generalisation der Aktinomykose, Virchow's Archiv. **255**: 719, 1925.
61. West, Samuel: Case of Primary Actinomycosis of the Pleura in a Child of Six With a Table and Analysis of Recorded Cases of Primary Actinomycosis of the Lung and Pleura, Tr. Path. Soc. London, **48**: 17, 1897.
62. Wildenburg, Louis: Deep Actinomycosis of the Neck and Mediastinum, Arch. Otolaryngol. **7**: 50, 1928.
63. Wilkinson, H. F.: Pathologic Studies in Tonsils—a Study of 10,000 Cases of Tonsils With Special Reference to the Presence of Cartilage, Bone, Tuberculosis and Bodies Suggestive of Actinomycosis, Arch. Otolaryng. **10**: 124, 1929.
64. Wolff, M., and Israel, J.: Ueber Reincultur des Actinomyces und seine Uebertragbarkeit auf Thiere, Virchow's Archiv. **126**: 11, 1891.
65. Wright, J. H.: The Biology of the Organism of Actinomycosis, J. Med. Research. **13**: 349, 1905.
66. Wynn: A Case of Actinomycosis: Streptotrichosis of the Lung and Liver Successfully Treated With a Vaccine, Brit. M. J. **1**: 554, 1908.

DISCUSSION

DR. FREDERICK T. LORD (BOSTON).—With respect to the portal of entry of actinomycetes into the body, there is much confusion in the literature. This is largely due to the investigations of Bostroem (Beitr. z. path. Anat. u. z. allg. Path. **9**: 1, 1890) who succeeded in isolating a branching, filamentous organism growing under aerobic conditions at room and incubator temperature from a small proportion of cultures from bovine and human actinomycosis. Lesions like actinomycosis were not reproduced in animals. Though Bostroem's organisms are with probability to be regarded as contaminations in his cultures, or secondary invaders in the lesions, the acceptance of his findings and the discovery of similar organisms widely distributed in nature has led to the conception that actinomyces may be implanted in the tissues through the medium of grains or other vegetable material.

An organism (Actinomyces bovis) described by Wolff and Israel (Virchow's Arch. **126**: 11, 1891), Wright (J. Med. Research, **13**: 349, 1905, Publication of the Massachusetts General Hospital, **1**: No. 1, May 1905), and others is to be regarded as the cause of actinomycosis. It is an anaerobic, nonspore-bearing, Gram-staining, branching, filamentous organism which grows well only at incubator temperature. Intraperitoneal inoculation of animals with pure cultures leads to the inconstant production of lesions histologically identical with typical actinomycosis, but without a progressive character comparable to the disease in man and cattle. These results suggest an additional and as yet unknown factor in the pathogenesis. The synergistic action of other organisms with Actinomyces bovis is to be considered, but evidence for this is thus far lacking.

The mode of infection has interested me for a number of years. Actinomycosis involves the head and neck in more than half the cases and its point of origin can often be traced to the tissue in or about the buccal and pharyngeal cavities. Trouble with the teeth may precede the onset and a sinus may be traced to the root canal of a carious tooth. The initial lesion when the disease involves the thorax or abdomen is usually in the lung or the gastrointestinal tract. Here, too, the buccal cavity may be regarded as the probable source of infection from inhaled or swallowed organisms. As the Actinomyces bovis grows well only at body temperature it is reasonable to assume that it must find its habitat in the human or animal body. Acting on this supposition, I (Lord: A Contribution to the Etiology of Actinomycosis. The Experimental Production of Actinomycosis in Guinea Pigs Inoculated With the Contents of Carious Teeth, Boston M. and S. J. **163**: 82-85, July 21, 1910; The Etiology of Actinomycosis: The Presence of Actinomycetes in the Contents of Carious Teeth and the Tonsillar Crypts of Patients Without Actinomycosis, J. A. M. A. Oct. 8, 1910; The Etiology of Actinomycosis. Proceedings of the Third Annual Meeting of the Am. Soc. for Advancement of Clinical Investigation, J. A. M. A. **57**: 148-151, July 8, 1911) found in the contents of carious teeth and the crypts of the tonsils of patients without actinomycosis organisms morphologically and by staining reaction identical with Actinomyces bovis. The intraperitoneal inoculation of guinea pigs with this material and the scrapings from sound teeth gave rise to omental tumors histologically identical with actinomycotic tissue and containing typical club-bearing actinomyces granules.

The proof, however, that this organism harbored in the normal mouth was Actinomyces bovis failed because I was unable to obtain pure cultures from the original material or from the animal lesions on account of the abundance of contaminating bacteria.

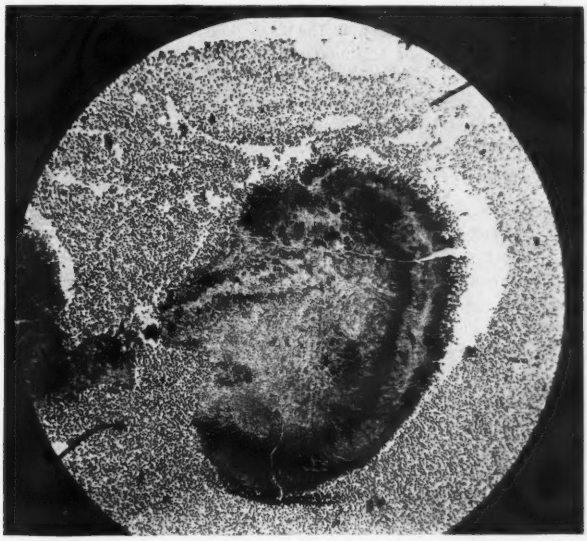
Further evidence in favor of the origin of actinomycosis from organisms harbored in the mouth by normal persons has, however, been obtained by Naeslund (Acta path. et microbiol. Scandinav. **2**: 110, 1925; Ibid, 1931 Suppl. VI) who in confirmation of my findings, produced typical actinomycotic lesions in animals inoculated with material from the contents of carious teeth, saliva, and dental scum. He succeeded, where I failed, in obtaining from the mouth cavity of normal persons, pure cultures of anaerobic organisms morphologically, culturally, and serologically similar to the actinomycetes of the Wolff-Israel type and capable of giving rise in animals to typical actinomycotic lesions with club-bearing granules.

As the matter now stands from the clinical aspects of the disease, the biology of the Actinomyces bovis and the experimental observations, actinomycosis is to be regarded as arising in consequence of organisms harbored in the mouth by normal persons.

DR. EVARTS A. GRAHAM (ST. LOUIS).—I think this has been a beautiful presentation by Dr. Wangensteen of this subject. I should like briefly to refer to

a patient who came under our care last fall, who apparently had pulmonary actinomycosis which was treated successfully by cautery pneumectomy.

The patient was a man, aged fifty-one years, a banker, referred by Dr. L. Behrens. He was admitted to the Barnes Hospital, September 28, 1931, complaining of a productive cough and fever of three weeks' duration. The onset was acute but the course was not at all typical of pneumonia. There was a suspicion of fluid in the lower right pleural cavity and aspiration resulted in the withdrawal of thin foul pus which contained fusiform bacilli. Because of a suspicion that the patient had a carcinoma of the lung some of the pus was saved for study by the Mandelbaum method. The leucocyte count was 27,800. A blood culture was negative. Aspirations of the pleural cavity were repeated on October 2 and October 4 at which times 600 c.c. and 300 c.c. respectively, of foul pus were aspirated. On October



Hematoxylin and eosin stain of sediment examined by Mandelbaum method. Typical colony of actinomyces.

5 closed drainage of the right pleural cavity was established. In spite of the profuse drainage of foul pus the patient continued to remain febrile. The pus which had previously been saved for examination by the Mandelbaum method was now found on microscopic section to show a typical colony of actinomyces. Accordingly, on October 10 the rib immediately below the drainage tract was resected to permit open drainage. A cavity containing 50 c.c. of very foul and moderately thick pus was entered. The cavity was roughly divided into two parts by a border of lung tissue which projected into it. There was nothing in the gross appearance of the pus which suggested actinomycosis. In spite of adequate drainage from the empyema cavity the patient's symptoms did not improve and in fact he gradually became more febrile and more toxic. It was apparent that probably his symptoms were coming from his pulmonary infection. Accordingly, on October 27 the old incision was enlarged and the two ribs immediately above were resected for a distance of about four inches. The roof of the empyema cavity was removed and a cautery pneumectomy

of the diseased portion of the lung was performed. A cavity slightly smaller than the patient's fist was produced with the cautery. The lung tissue everywhere was necrotic and resembled a sponge soaked in pus. There was no evidence of a carcinoma and no sulphur bodies were seen. A piece of lung tissue removed for examination showed nothing but inflammation and chronic pleuritis. There was nothing in the lung tissue suggestive of actinomycosis. Deep x-ray treatments were also given through the open wound and, at the suggestion of the family physician, the dressings applied to the wound were soaked in staphylococcus aureus bacteriophage. About two weeks after the operation the patient developed weakness in the right arm which aroused a suspicion of a cerebral metastasis but no definite evidence of such a lesion could be obtained and the patient from that time on made a rapid convalescence and at the present time has been entirely healed and free from all symptoms for a period of three months. He seems to be entirely well.

The principal point about this case, which makes one hesitate to call it definitely a case of pulmonary actinomycosis, is the fact that on only one occasion were we able to find a typical colony of actinomyces and at no time did we see any sulphur granules, although careful search was made for them in the pus from the wound. On the other hand, it is difficult to see how any contamination with this organism could have occurred because it was found in the aspirated pleural fluid using the ordinary aseptic precautions.

DR. CARL A. HEDBLOM (CHICAGO).—I should like to comment on Dr. Wangensteen's statement that different strains of organisms that present the characteristic morphology of Actinomyces bovis differ in their effect on the organism.

A patient under my observation at the Research Hospital of the University of Illinois, about two months ago seems a case in point. The patient was a male musician sixty years of age, who had had increasing pain in the chest with cough and purulent sputum for four months. During this period he had lost fifty pounds in weight. A few weeks before admission he discovered a mass on the left anterior chest wall.

On examination an indefinite fluctuating mass was felt in the left anterior second interspace and roentgenologic examination showed a sharply outlined shadow about the size of a medium-sized orange, with destruction of part of the third rib. The sputum showed the typical club-shaped organisms of actinomycosis. Pus aspirated from the tumor was sterile to culture and no organisms were found on microscopic examination of the smear.

Exploratory operation revealed an abscess situated superficial to the parietal pleura, with extensive destruction of the third rib and a curious tawny, greyish, dense induration of the parietal pleura forming the bed of the abscess.

The patient died suddenly a few days after operation with symptoms of myocardial failure. Necropsy showed a carcinoma of the lung. There was no evidence of actinomycotic lesion of the lung.

The point I wish to make is that the symptoms in this case were due to carcinoma and that we were misled by the morphologic appearance of club-shaped organisms in the sputum.

One case of true actinomycosis of the chest wall which I treated successfully followed a scratch of the skin over the sternum inflicted by a playful cat. The patient developed the typical extensive infection with multiple sinus formation which persisted for several years but which finally cleared up after extensive rib resection. Later she developed a sinus in the loin.

Several cases of actinomycosis under my care followed appendectomy for what seemed to be ordinary appendicitis. The first manifestation of the actinomycosis was sinus formation in the scar from which actinomycosis organisms were demon-

strated. In several of these cases wherein complications developed later an empyema of such origin was cured by prolonged open drainage.

DR. JOHN ALEXANDER (UNIVERSITY HOSPITAL, ANN ARBOR, MICHIGAN).—Perhaps actinomycosis occurs more frequently in the Middle West than in the East. I have seen a number of cases here, including four or five involving the thorax, but none that I can recall in Philadelphia.

When actinomycosis cannot be certainly diagnosed by smear or culture, a strong presumption of the disease exists if during an operation the indurated tissue bleeds more than one would expect tuberculous or the usual pyogenic tissue to bleed and if a specimen of tissue removed for microscopic examination shows fatty degeneration of the many leucocytes and mononuclear phagocytic cells and a great many thin-walled blood vessels.

Among the four or five thoracic cases that I have treated by wide excisions or mere drainages, there has been no cure. The disease has been slow in its progress and has extended widely. All of the patients were greatly relieved by adequate drainage of the large and medium-sized abscesses. Although a high temperature may become normal and the general condition be much improved, the pulse tends to remain very high.

DR. FREDERICK T. LORD.—Regarding the diagnosis from the examination of material from the lesion, cases may with probability be classified as infection with Actinomyces bovis when granules composed of a dense mass of gram-staining, branching filaments with radially disposed, club-shaped bodies with central filament can be found in the exudate or the tissue. An origin of the disease from the alimentary canal and especially the cervicofacial type is still further suggestive. Actinomyces bovis infection may with probability be differentiated from that due to the streptothrix group by the presence in the latter of loose clusters of acid and alcohol-fast, gram-staining filaments, at times with club-shaped extremities. An origin of the infection in the lung with or without metastases, especially to the brain, is suggestive of streptothrix invasion. Though radiate structure of the colonies and the formation of true clubs are specially characteristic of infection with Actinomyces bovis, recent observations by Naeslund indicate that pathogenic, aerobic organisms of the streptothrix group are capable of producing in inoculated animals more or less similar colonies. For complete identification of the etiologic agent, therefore, observation of the cultural characters and the results of animal inoculation are desirable. Agglutination by Naeslung of anaerobic and aerobic organisms by homologous and heterologous antisera in high dilution suggests a close relationship between the two groups.

DR. RICHARD H. MEADE, JR. (PHILADELPHIA, PA.).—I should like to report a case of actinomycosis of the thorax that I have been following for four years. The patient was a forty-seven-year-old white farmer who entered the University of Virginia Hospital in April, 1928 with diabetes and an empyema with a bronchial fistula.

Aspiration of the pleural cavity yielded pus from which B. proteus was grown. Drainage was established by rib resection, greatly thickened pleura being encountered. After a few weeks an abscess developed in the chest wall below the pleural drainage wound and was incised. No organisms could be cultured from the pus. Later, more adequate drainage of the empyema cavity was accomplished and examination of the thickened pleura removed showed only chronic inflammatory tissue with no bacteria. A sluggish abscess undermined the corresponding portion of the abdominal wall and when drained also failed to reveal organisms.

Five months after admission the typical sulphur granules of actinomycosis appeared in great profusion in the pus of all wounds. Massive iodine administration

was then started. During the next month an old sinus in the angle of the left jaw discharged actinomycotic pus and a new abscess was drained in the left axilla. Following treatment by means of iodine, roentgen ray, surgery, insulin, and diet, he left the hospital in June, 1929 in good condition except for a draining sinus in his chest and in his abdominal wall.

He was entirely well from November, 1929 until October, 1930, when he was readmitted to the hospital because of an infected foot and diabetes. At this time no evidence of actinomycosis could be found, and roentgen ray examination of his chest showed only a thickening of the pleura.

In September, 1931, twenty-two months after his last wound had healed, he again began to cough and had slight discharge from his chest wound. A recent report says that he is coughing up bloody mucus and a roentgenogram of his chest suggests a recurrence of the disease in his lung.

DR. OWEN H. WANGENSTEEN (MINNEAPOLIS).—It is indeed refreshing to note the lively interest in this rather discouraging subject. Dr. Lord's studies concerning the mode of invasion in actinomycosis are well known and I am grateful to him for his discussion. The most intriguing suggestion that actinomycosis may arise from organisms within the mouth is to be found in the case reports of Zachary Cope of London and of the late McWilliams of New York, each of whom has reported an instance of actinomycosis developing on the finger following an injury sustained on an adversary's tooth.

In the case reported by Dr. Graham, I feel it cannot be denied that this must be actinomycosis, unless one can satisfactorily account for the presence of the actinomyces in the lesion otherwise, such as a lung abscess with a bronchial communication. Though streptothrix organisms are commonly found in the sputa of many types of pulmonary suppuration, as shown by Thompson, these organisms demonstrate no tendency to invade the pulmonary tissues. It is somewhat strange that no sulphur granules were found in the pulmonary tissues or chest wall.

There is one point relative to actinomycosis that I should like to stress; viz., that it exhibits an unusual tendency to spontaneous disappearance at the portal of entry. This is equally as true of cervicofacial and abdominal actinomycosis as it is of the pulmonary variety. One must not be discouraged therefore by the seemingly formidable task of dealing with actinomycosis of lung and chest wall in a patient who is a bad risk for a major undertaking. Incision and drainage oftentimes permit the patient to improve considerably; radical excision should only be done when simpler methods alone are inadequate.

I feel definitely that if we will buckle up our courage in the management of pulmonary and abdominal actinomycosis that there will be many more successes. Walter Brickner of New York has reported a number of instances of abdominal actinomycosis that he has successfully treated by incision, excision, and drainage.

The thought that I would like to leave with you is this: We can cure these cases, but we must not rely upon the conservative agents of x-ray and potassium iodide. They are futile remedies in thoracic actinomycosis and hold out no hope to patients so afflicted.

PUTRID LUNG ABSCESS—ITS ETIOLOGY, PATHOLOGY, CLINICAL MANIFESTATIONS, DIAGNOSIS AND TREATMENT*

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THE results of a joint study of lung abscess, made by a number of coworkers and ourselves at the Mount Sinai Hospital, are summarized in this paper. Contributions from the members of the group will be forthcoming, amplifying the statements we shall make. Clinically, abscess of the lung is practically synonymous with putrid abscess of the lung. Nonputrid varieties, such as those due to the staphylococcus or pneumococcus, present clinical manifestations referable chiefly to invasion of the pleura and will not be discussed, for this contribution deals solely with the only type of lung abscess that is commonly encountered as a clinical condition. Putrid empyema, which is an important complication, is omitted.

There is no subject in clinical medicine whose literature presents greater diversity of views concerning etiology, pathology, and treatment. Lung abscess is far from rare, is usually serious, and is often fatal in the subacute and chronic stages. It is frequently confused with bronchiectasis as well as with other bronchopulmonary lesions. Our studies have aided us greatly in an understanding of its nature and have proved of definite value in its diagnosis and treatment. A summary of our views and experiences is therefore warranted at this time.

ETIOLOGY AND PATHOGENESIS

Putrid lung abscess is a definite clinical and pathologic entity. The infection is probably initiated and is certainly maintained by pathogenic anaerobes. It is of bronchogenic origin, and is due to aspiration of infective material. In our series of hundreds of cases there were only a very few instances in which the lesion could be interpreted as being embolic or probably embolic in nature. These rare exceptions presented clinical manifestations and pathologic changes quite different from those to be described in this paper. Experimentally, embolic lung abscesses can be readily produced but lung abscess via the bronchi, only with difficulty. This observation among others has led many to assume that human lung abscess is often of embolic origin. The fact is that experimental embolic lung abscess in no way resembles putrid lung abscess in

*From the Surgical and Medical Services and the Laboratories of the Mount Sinai Hospital.
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human beings. The mechanism and the characteristics of embolic abscesses in the human body have been amply demonstrated. Embolic abscesses are derived from a septic focus, usually in a vein, are ordinarily multiple, and are scattered in various parts of the pulmonary parenchyma. They only rarely and then only accidentally communicate with bronchi, are a silent feature of sepsis in most instances except for pleural invasion, are usually due to the staphylococcus or streptococcus and are thus nonfetid. These and other features are in striking contrast to those of putrid lung abscess as will be shown. The question is of clinical as well as of scientific interest, for a belief in the embolic origin and pathology of putrid lung abscess would lead to a different plan of treatment from that to be outlined.

We believe that putrid lung abscess results from the aspiration of infective particulate material bearing pathogenic anaerobic bacteria. The concept of the disease solely as a sequel to operations on the nose, mouth, or throat, or to operations under inhalation anesthesia, or to known aspiration of foreign material, is erroneous. About one-third of the cases in our series of the past two years occurred in previously healthy persons who presented no proved predisposing factor. The concept of putrid lung abscess as a complication of pneumonia is also incorrect. Our evidence is clear that the disease is not a complication of pneumonia, but that the so-called pneumonia was a putrid lung abscess from the onset. This is a matter of no little clinical significance, for the diagnosis can be made in the stage of so-called pneumonia in many instances.

Long before conclusive bacteriologic studies were made we with others believed that putrid lung abscess was an anaerobic infection. The clinical characteristics were those of an anaerobic infection and observation of the results of surgical treatment based on that view tended to corroborate our belief. We can now state that appropriate bacteriologic study of pus obtained at the time of operation has confirmed our clinical impression. Certain pathogenic anaerobes are to be found in every stage of the infection and disappear when the infection subsides. There is no absolute evidence that the disease is initiated by these anaerobes. They are, however, the only pathogenic organisms found in putrid lung abscess which produce gangrenous lesions of the lungs experimentally when introduced into the bronchial tree.

PATHOLOGY

Putrid lung abscess characteristically begins in and distal to, one of the smaller bronchi at the site at which the aspirated infective material is presumably arrested. A suppurative process develops that is unique in human pathology. The following outline is based on numerous observations at operation and at autopsy. An intense necrotizing inflammation of the affected bronchus and its tributary bronchioles is the first

stage. The pulmonary parenchyma to which the involved bronchioles are distributed soon becomes included in the same severe inflammatory process. The lesion is thus always situated near the surface of the lung, and a pronounced and early reaction of the overlying pleura invariably occurs. Rapid destruction of bronchial walls and blood vessels takes place in the involved area. A localized gangrenous abscess superficially situated in the lung and usually of large size is the result. The cavity contains foul pus and detritus, and liquefying sloughs of lung. Its interior becomes smooth-walled at an early stage. Opening into the cavity are the patent mouths of one or more bronchi of the smaller order. Thrombosed vessels are to be noted on or in the walls. Drainage by way of the bronchus (expectoration of foul pus) usually begins within two weeks of the onset. Thereafter the course depends largely on the degrees of bronchial drainage. A wide communication of the abscess with a bronchus will permit sloughs to escape from and air to enter the cavity and will thereby favor healing. If drainage and ventilation by way of the bronchus are incomplete, the pathologic process will persist. Exceptionally it rapidly extends directly and by spill-over into other bronchi, to produce the pathologic picture of a rapidly fatal gangrenous bronchopneumonia. In other cases there is progressive necrosis of the parenchyma with rupture into the pleural cavity in the zone of adhesions. In the majority of instances, however, the abscess either remains localized and more or less quiescent for weeks or months with occasional spreads, or insidiously involves the adjoining lung in this subacute phase. In either event early fibrosis of the adjacent parenchyma is a striking and unique feature. As stated, the spread may be by direct extension of the gangrenous process, but a more significant extension is by spill-over of the putrid material from the involved bronchus to adjoining bronchi. The result is a multilocular abscess, surrounded by fibrotic lung, as the chronic phase is reached in from two to four months after the onset. Healing by bronchial drainage (spontaneous healing) rarely occurs after the chronic phase begins. Dilatation of the bronchioles around the abscess (localized bronchiolectasis) often sets in, even in the subacute phase, but is no more distinctive of putrid lung abscess than of other inflammatory processes in the lungs. As the lung abscess becomes more definitively chronic, however, the dilatation of bronchioles and bronchi usually becomes more evident and diffuse, and may finally overshadow the original lesion. It is for this reason that putrid lung abscess and bronchiectasis are so commonly confused, both pathologically and clinically. Furthermore, in the later stages the dilated bronchi may become secondarily infected by spill-over of pus from the lung abscess, so that a putrid bronchiectasis will result and the original lesion may be found only by very careful search. The last phase of the pathology of lung abscess, in which multiple involvement by spill-over and by direct extension

exists, and in which amyloidosis or other complications usher in the end, is merely mentioned here as the irremediable stage.

In addition to the usual place of onset of the pathologic process in a small-sized bronchus (third, fourth, or fifth order), two other sites may be noted. The first is a large bronchus in which a foreign body, such as an aspirated tooth, becomes lodged. The typical pathology of putrid lung abscess will not be seen. The second site is a terminal bronchiole and a cortical putrid lung abscess will then result. The lesion is on a much smaller scale and prompt recovery by drainage through the bronchiole undoubtedly occurs in many cases which are not often recognized clinically. On the other hand, the subpleural situation of such an abscess renders rupture into the pleura likely if drainage through the bronchiole is inadequate or lacking. The resultant putrid empyema or pyopneumothorax is usually limited by firm adhesions.

CLINICAL MANIFESTATIONS

Although the later clinical manifestations of lung abscess may be bizarre, there is usually an orderly sequence of events in the initial stage. The interval of time between aspiration and the onset of symptoms is of course known only in those cases in which the etiology is known. A definite period of incubation, in which there are no obvious symptoms, can be said to exist. It is generally of three or four days' duration. The initial symptoms are feverishness and chilliness or an actual chill. They are shortly followed by pain in the chest, a constant phenomenon, usually severe and sharply localized. Supradiaphragmatic abscesses (with pain referred to the shoulder) and abscesses facing the interlobar fissures comprise rare exceptions. A matter of great surgical significance is the fact that the site of localized pain is identical with the site of pleural adhesions. Circumscribed tenderness to pressure is associated with the pain. Pain lessens after the first few days, and may have to be recalled to mind in a patient who has reached the chronic stage. Cough usually begins after the onset of pain and is unproductive at first. A foul odor on coughing may be noted. Expectoration of foul pus begins suddenly, and at a characteristic time in the great majority of cases. This is the tenth to twelfth day after the onset. If not truly foul and copious during the later stages, the sputum is always frankly stinking and usually profuse in the beginning. Early hemoptysis, varying from blood streaking to active hemorrhage, is never lacking. It is therefore a commoner symptom of lung abscess than of tuberculosis. Although foul odor on coughing may be present, the expectoration of foul pus does not occur (1) in the small cortical type of abscess, and (2) in exceptional instances in which none of the contents of the abscess can escape through the bronchus (shut-off abscesses).

The further course is usually in one of three directions. First, the de-

velopment of a septic state, accompanied by the expectoration of pus that becomes progressively more foul. This is a rare type which usually goes on to an early fatal termination. The second form is subsidence and early cure, probably dependent on wide bronchial communication. The third is the usual course. In a typical case, relief follows the expectoration of the pus which is often brought up in large amounts. Fever and pain subside. Improvement appears to be progressive for a period of from two to four weeks. Recovery may be actual, with the disappearance of foul sputum and the other evidences of abscess which will be noted later. With rare exceptions, recovery does not occur after the first two or three months. Spontaneous cure occurred in about 15 per cent of the cases in one of our series and in 30 per cent in another series. Many patients appear to recover, only to pass into the subacute and chronic stages.

The subacute phase is characterized by the appearance of clubbing of fingers and toes (also to be noted in the acute stage), by recurrence of fetid expectoration and fever, pain in the chest, and roentgen ray evidences of spread of the infection. In other instances the lung abscess is shut off and the expectoration may continue but remain nonfetid indefinitely. Under such circumstances copious nonfetid expectoration indicates a bronchiectasis around a shut-off abscess.

Only three of the varied manifestations of the chronic stage will be mentioned. In one the patient remains in fair comfort for a long time except for the expectoration of foul pus. In the second there are recurrent attacks of fever and other evidences of spread of the infection. In the third variety, the anaerobic infection is more or less burned out, foul sputum is rarely present, and the manifestations are essentially those of bronchiectasis. Whatever the evolution of symptoms, the course of a confirmed chronic lung abscess is a progressively downhill one, with extension of the disease to adjacent and distant parts of the lung by direct invasion and by spill-over. If untreated or inadequately treated, the disease is almost invariably fatal, death occurring from pulmonary complications or from cerebral or other metastases. In adults death usually takes place within three years of the onset, and in a much shorter time in the great majority of cases. Unless early spontaneous recovery occurs, lung abscess is almost as fatal as cancer and usually kills more quickly.

DIAGNOSIS: ROENTGENOGRAPHY

The diagnosis can usually be made on the history and a careful survey of the clinical manifestations. It cannot be established by physical signs alone. The latter are inconstant, being slight or absent in some instances and equivocal in others. When present, the signs consist essentially in a more or less localized area of dullness over which the breath sounds and voice are diminished. Signs of cavitation are rare.

Reference has already been made to localized tenderness to pressure. Any diagnostic value of these noncharacteristic physical signs lies in their insignificance in the presence of an otherwise obvious pulmonary lesion. The physical signs attending rupture of an abscess into the pleura are those of any empyema or pyopneumothorax.

The roentgenogram establishes or confirms the diagnosis in the majority of cases. The well known film of cavity with fluid level is pathognomonic although the differentiation between lung abscess and pyopneumothorax cannot be made in some instances. The shadow may occupy any part of the pulmonary field. There are certain areas of predilection definitely related to the pulmonary segments to which third and fourth order bronchi are distributed. Details cannot be given, but one important fact can be stated: mesially situated shadows invariably indicate lung abscesses occupying the posterior, paravertebral parts of the lung.

Typical films of lung abscess were encountered in no more than one-half of our cases. The remaining half fall into a number of groups, of which the following are selected because of clinical significance: (1) Film of pneumonitis. An abscess approximating the size of the shadow may in fact be present, and the assumed pneumonitis prove to be the undrained abscess. We have followed a number of cases of lung abscess in which a fluid level was never seen in the area of supposed pneumonitis despite repeated x-rays taken over periods of many months. (2) Film of pneumonitis with small fluid level, ordinarily interpreted as a small abscess with surrounding pneumonia. Most of these cases are actually large abscesses with limited encircling pulmonary infiltration. The presence or absence of a fluid level depends on the accident of good or insufficient drainage through the bronchus. (3) Films of apparent variation in the size of an abscess are often deceptive. Apparent diminution in size is no evidence of improvement unless accompanied by diminution in the extent of the enveloping infiltration. (4) Disappearance of cavity does not mean cure unless associated with disappearance of infiltration. Disappearance of cavity with increase of infiltration not only signifies persistence of the abscess, but also the unlikelihood of spontaneous cure. (5) The film of fibrosis in chronic lung abscess. Cavities may not be recognizable as such. The films of the earlier stage must be studied in order to identify the site of the abscess in the midst of the fibrotic area. (6) The film pointing to centrally situated abscess. This may be the only possible localization from the roentgenogram. The lesion is either not a lung abscess or the localization is incorrect, because lung abscesses are always near the surface. (7) The film of apparently multiple abscesses. In most instances the abscess is multilocular and not multiple despite the appearance of completely separated foci. (8) The film of chronic lung abscess may closely simulate that of fibroid

tuberculosis. In addition, tuberculosis may be present in one part of the lung and an abscess in another. Furthermore, a putrid lung abscess occasionally occurs in the midst of unrecognized pulmonary tuberculosis. The latter may not be recognizable because we have not, up to the present, found tubercle bacilli in the sputum while the putrid infection exists in such cases. (9) The film may be strongly suggestive of carcinoma and not indicative of lung abscess. The differentiation can only be made by the history, general examination, and bronchoscopy.

DIAGNOSIS: BRONCHOSCOPY AND BRONCHOGRAPHY

Bronchoscopic examination is indicated in every case of subacute and chronic lung abscess and in most instances of acute lung abscess. It establishes the existence or absence of foreign bodies, benign or malignant tumors, bronchiectasis, etc. The presence of a single or multiple source of foul pus can be determined. Bronchoscopy often discloses the precise source of the pus and therefore aids in determining the exact situation of the abscess. The examination is particularly valuable when there is no expectoration of foul pus. Under these conditions a foul odor or foul pus may be detected through the bronchoscope. In our series there were a number of instances in which a lung abscess was barely suspected and in which the bronchoscopic examination established its presence. Unless improper force is used, the bronchoscope cannot enter a putrid lung abscess (see pathology). There are very rare exceptions to this statement.

Bronchography by the use of iodized oil is an invaluable method for localization of the abscess, especially in difficult problems and in patients already operated upon and not making satisfactory progress. Our bronchographic examinaton, done under fluoroscopic inspection, is selective, the purpose being to fill only that portion of the bronchial tree related to the site of the abscess. A knowledge of the anatomy of the bronchial tree is essential, particularly of the segmental distribution of the branches of the third and fourth order. The iodized oil is no more capable of entering the abscess cavity than is the bronchoscope. The bronchogram of lung abscess is therefore an outline of all the branches of the bronchial tree with the exception of the one connected with the abscess. It also demonstrates the presence and extent, or the absence of bronchiectasis. Precise localization of the lesion for purposes of surgical approach has been afforded by bronchography in many cases in our series.

TREATMENT

Putrid lung abscess is potentially a surgical lesion from the outset. There is no proved method of definitively influencing the course other than by open operation. Drugs are of no value except for the relief of pain and discomfort. Postural drainage aids in the evacuation of some

of the contents of the abscess and is therefore useful to that extent in acute cases with profuse expectoration. Phrenicectomy may possibly invite sufficient relaxation to assist in the healing of an acute abscess, but is of no proved value because spontaneous healing may occur in the acute phase. The same statement can be applied to pneumothorax. Pneumothorax, however, can only be effective by breaking down the adhesions situated directly over the superficially placed abscess (see pathology). The sequel may be a fulminating, putrid, fatal empyema, of which we have seen several instances. Pneumothorax is too dangerous and too dubious a procedure to have any place in the treatment of lung abscess.

Bronchoscopy is essentially a diagnostic method in lung abscess. The removal of granulations from the involved bronchus may aid in the drainage of an acute or subacute lung abscess, and if found useful for this purpose, can be repeated. Nothing more than improvement in drainage can be anticipated from the bronchoscopic treatment of chronic lung abscess in the great majority of cases. That bronchoscopic treatment by instillations or irrigations cannot cure chronic lung abscess is evident from a knowledge of the pathology of the disease. Reports in the literature of high percentages of cures must refer to other conditions. Pioneer work in the bronchoscopic treatment of lung abscess was done at the Mount Sinai Hospital by Yankauer, yet the method is not practiced there at the present time.

Of the various operative procedures that have been advocated, thoracoplasty appears to have the greatest vogue. That the operation is illogical is evident from the outline of the pathology and bacteriology which we have given. Open drainage was the earliest operation for lung abscess. High mortality and frequent failure to cure, led to more radical procedures culminating in lobectomy. The latter by any technic is formidable, but usually curative when patients recover from operation. We reserve it for exceptional cases.

The principles of the operation we practice apply to resistant abscesses elsewhere in the body, with special attention to the factor of anaerobic infection. These principles are excision of the roof and ventilation. The operation cannot be termed incision and drainage, which was the original procedure for lung abscess so frequently followed by failure.

Acute lung abscess is generally looked upon as a pneumonia with scattered areas of gangrene in its midst and therefore obviously not a surgical condition. We consider acute lung abscess a surgical lesion, basing our view on (1) the described pathology of the lesion, (2) the disclosure of overlying adhesions and a localized abscess in the lung at operation, and (3) the results of operation. Operation in the acute phase is safer than in the chronic phase and the results as to definitive cure are far better. Operative treatment of acute lung abscess is indicated if there is an increase in size under observation or if the clinical

course points to the unlikelihood of spontaneous cure. The exception is the rare and usually rapidly fatal type with spillover infection and gangrenous bronchopneumonia. Operation is always indicated for subacute abscess. It must be deferred in the presence of an acute gangrenous extension. Operation is the treatment of chronic lung abscess unless in the irremediable stage (see pathology).

The site of operative approach should be based on precise information derived from the diagnostic measures that have been enumerated. The greatest danger of operation is the development of putrid empyema resulting from entry of the free pleural space. The abscess should be entered only through the zone of pleural adhesions. If no adhesions or insignificant adhesions are encountered, the approach is incorrect. The correct site must be found either at that time or at a later operation. Partial excision of one rib usually suffices. The abscess is sought for by aspirating through the thickened pleura. Only a thin shell of infiltrated lung will be traversed in the majority of cases before foul pus and detritus are encountered. If the abscess is first encountered at a depth of 5 cm. or more, the site of aspiration is probably an incorrect guide for incision of the abscess. The aspirating needle readily discloses the abscess in most acute and subacute cases. In the chronic cases, however, many aspirations through the fibrotic lung may be required. When the abscess is found, its roof is split open and later excised. It is usually larger than had been anticipated before operation, from 5 to 7 cm. being average diameters. After evacuation of the contents, the interior is inspected for bronchial openings and for recesses. There are usually few difficulties at operations for acute or subacute abscess, the lesion being essentially single and the identification of structures easy. The unroofing of all the ramifications of a chronic abscess may be exceedingly difficult, or even impossible at one sitting. The free opening of an abscess and the visualization of bronchial orifices does not constitute an adequate operation even though it proves to suffice in a given case. The operation can only be considered completed when all recesses from and communications with the main cavity have been laid open. In chronic abscess the ramifications may be more extensive than the original lesion. The abscess cavity tends to narrow quickly after operation and should therefore be widely packed at the close of operation.

The postoperative care of the patient and of the wound, and the management of complications are vital matters to which only passing reference can be made. The expectoration of foul pus ceases promptly after an adequate operation. The wound should be healthy-looking and free from odor within a week. Regardless of whether or not there was a known opening of the free pleura at operation, a putrid empyema should be sought for if a septic course exists after operation. The continuation of nonfetid expectoration after operation for chronic lung abscess may

be due to bronchiectasis or to the existence of an undrained abscess. The bronchial fistula or fistulae are maintained until all traces of anaerobic infection have disappeared and pulmonary infiltration has subsided. Permanent fistulae may be necessary in cases of long-standing chronic lung abscess. A patient can be termed well of a lung abscess only when there is lasting evidence of disappearance of pulmonary infiltration and of anaerobic (putrid) infection.

Detailed data of the results of treatment, carried out according to the principles we have outlined, cannot be given here. We shall confine ourselves to a brief analysis of known results in 100 unselected cases of putrid lung abscess observed in 1930 and 1931. From this series we exclude 24 cases of ruptured lung abscess, 4 cases associated with tuberculosis, and 3 cases of extension from the mediastinum. There were 20 patients with *acute lung abscess.* Ten were not operated upon because the clinical course was favorable, and all are well. One patient with the acute fulminating type died. Nine were operated upon because of the belief that spontaneous recovery was improbable. Of these 8 are well and one died. There were 8 patients with *subacute lung abscess.* Two with favorable clinical courses were unoperated; one is well and one is improved. Of the 6 patients operated upon three are well and three died. All 7 patients with *subacute lung abscess complicated by acute gangrenous extension* died whether operated upon or not. There were 34 cases of *chronic lung abscess.* Of the 13 patients not operated upon, 3 are improved, 4 are unimproved, 6 died. Twenty-one patients were operated upon. Of these 12 are well, 2 improved, 7 died.

Comparing the 1930 and 1931 cases, there is a higher incidence of cure and lower operative mortality in the 1931 series, largely referable to greater precision in localization and more frequent operations for acute lung abscess.

DISCUSSION

DR. FREDERICK T. LORD (BOSTON).—In connection with D. T. Smith's investigation on the symbiotic action of a group of anaerobic organisms, including spirochetes, fusiform bacilli, vibrios, and cocci as a cause of lung abscess, it may be noted that evidence is accumulating which suggests that at times in the vegetable world and in man bacterial association may be important. Meleny (Ann. Surg. **94**: 961, 1931) has reviewed the literature and reports instances of gangrene of the abdominal wall apparently due to the synergistic action of several organisms.

Dr. Neuhof has called attention to the usual peripheral site of lung abscess. Proximity to the pleura is in general a striking feature in cases coming to autopsy at the Massachusetts General Hospital and this may well be taken into consideration in connection with other aspects of the problem in the decision regarding operative treatment.

DR. CARL A. HEDBLOM (CHICAGO).—I endorse all that Dr. Neuhof has said regarding etiology, diagnosis, and treatment of pulmonary abscess. My own observations have led me to believe that the great majority of cases that occur as com-

plications of operative procedures, especially about the upper respiratory tract, are aspiratory in origin. I am also of the opinion that aspiration of infected material from pyorrhea alveolaris accounts for a large proportion of pulmonary abscesses that develop without obvious cause. Over 20 per cent of the cases in my experience belong to this group of uncertain or unknown etiology.

I was glad to hear Dr. Neuhof discuss the limitations and dangers of therapeutic pneumothorax and bronchoscopic lavage. Inasmuch as most pulmonary abscesses are peripheral there is always danger of empyema from tearing of adhesions, or from later perforation of an abscess. If this complication does not develop spontaneously, it is sure to do so if drainage is attempted while the lung is collapsed. In my opinion the chief value of bronchoscopy is in diagnosis and for improving drainage in centrally situated abscesses by removing etiologic foreign material or by dilating strictures. Many cases reported as cured by pneumothorax or by bronchoscopic lavage would probably have recovered spontaneously. The fundamental principle of treatment of pulmonary abscess is drainage, through the bronchus if centrally situated, and by thoracotomy if peripheral. Expectant treatment may be continued indefinitely if the patient improves progressively. If the disease process is at a standstill, and there are marked symptoms, drainage should be instituted before the destructive changes of chronicity have developed. If the progress is downward, unduly prolonged expectant treatment robs the patient of his chance for a cure by surgical drainage. The high mortality in chronic abscess is attributable to the pathologic changes brought about by ill advised "conservative" treatment rather than to the drainage operation as such.

A factor of safety of great importance in the drainage of an abscess is the use of the cautery, in opening the abscess. If spirochetes and fusiform bacilli infection are present it effectively prevents their metastases. I have lost three patients as a result of metastasis. Cautery also minimizes the danger of air embolism and effects better ultimate drainage than is achieved by tearing or cutting the lung parenchyma.

DR. J. J. SINGER (ST. LOUIS).—I do not believe that one can separate the treatment of pulmonary abscess into medical and surgical. I think the point of view of the surgeon, using the differentiation rather closely, is that any abscess ought to be opened or drained; I am heartily in favor of Dr. Hedblom's statement that if bronchial drainage is doing the work, why do more.

The fact remains that there are a large number of lung abscesses, not speaking particularly of the putrid type but abscesses in general, that do lend themselves, I won't say to medical treatment but to time, irrespective of what one does. In my experience at St. Louis I have seen a great number of patients with large abscesses who have recovered as a result of time, postural drainage, and the use of some of the arsenicals.

In regard to pneumothorax, I would be inclined to differ with Dr. Neuhof. There are a certain number of cases which do lend themselves to pneumothorax treatment but not to the type of pneumothorax that he is probably thinking about. This is an attempt to collapse the lung completely. For the same reason that pneumothorax as a partial collapse of the lung is used in tuberculosis, we have used it in certain types of lung abscess, particularly in those that seem to be away from the chest wall, as indicated by the x-rays. We have had a number of patients who have gotten well within three or four weeks after the introduction of a very small amount of air, merely enough to put the lung at rest and facilitate the drainage but with no attempt at collapsing the lung.

I do not believe that the lung abscess problem is at all settled. I think many other methods of treatment will be recommended.

DR. DEAN B. COLE (RICHMOND, VIRGINIA).—I wish to emphasize that the treatment of acute lung abscess is primarily medical and seldom surgical. In treating some 125 patients with lung abscess, we have found that most patients with acute abscess will recover without any treatment, other than bed rest and postural drainage. I also desire to substantiate Dr. Singer's experience with artificial pneumothorax in selected cases. While this procedure is seldom necessary, it may be employed with relative safety in any acute abscess which is draining into a bronchus, provided very small quantities of air, not more than 75 to 150 c.c. are given, as indicated. This produces a relaxation and not a compressive pneumothorax. It can be utilized regardless of the location of the abscess, but no attempt should be made to collapse the abscess cavity, but merely to squeeze gently the surrounding inflammatory tissue. The same principles are applied whether the abscesses are single or multiple. Gangrene of the lung responds poorly to all treatment, but some of these patients are apparently benefited by iodized oil therapy.

DR. CHARLES D. LOCKWOOD (PASADENA, CALIFORNIA).—I believe embolism plays an important rôle in the causation of putrid lung abscess.

We had one case which it seems to me was very definitely the result of embolic infection. The patient suffered compound fracture of the tibia. On the fourth or fifth day she was seized with severe pain in her left upper chest, and a sharp rise in temperature. She began to cough and the lung gradually broke down with a very putrid sputum. She was an old woman and her general condition was so poor that it was impossible to do anything surgically for her.

On postmortem we found a large thrombus involving the femoral vein, extending as high up as the external iliac. There the thrombus had degenerated and an embolus had been carried to one of the main branches of the pulmonary artery in the upper lobe of the left lung. Following the embolic infection a large abscess had formed, as large as a hen's egg, and from this she died.

Smears from the thrombus in the femoral vein showed a large number of gram-positive staphylococci. Smears from the lung in the involved left lobe showed similar organisms. There was some secondary infection, but the predominant infection was identical with the infection in the thrombus.

DR. HAROLD NEUHOF (NEW YORK CITY).—I should like to state that within the past two weeks there was a tragic case of lung abscess treated by low tension pneumothorax, followed immediately by a virulent putrid empyema, which resulted in the patient's death. I have seen at least six examples of such fatal putrid empyema, not only after the type of collapse pneumothorax to which two of the discussors referred but also after low tension pneumothorax. In the autopsy upon the patient to whom I referred there was a small, well-localized, superficially-placed lung abscess, with delicate adhesions around its periphery and denser adhesions at its center.

Unless there is any evidence—and as far as I am able to gather up to the present time there is no evidence—of cure as a result of pneumothorax treatment of acute lung abscess, I believe that it should be abandoned. It is entirely irrational to anticipate cure in chronic cases and, as has been stated, the incidence of spontaneous cure in acute cases is such that one is quite unable to determine whether in the instance in which pneumothorax has been applied the treatment by pneumothorax has resulted in cure, or spontaneous cure has occurred.

In the brief time at my disposal, I should like to call attention anew to the fact that in follow-up, on a large series of cases, no more than 15 to 30 per cent recovered spontaneously in the acute phase. That is, there is frequently enough apparent recovery, and then these cases go on to the subacute and more particularly to the chronic phase, in which they are so frequently classified as cases of bronchiectasis or putrid bronchiectasis, or other pathologic processes.

DR. CONRAD GEORG, JR. (ANN ARBOR, MICHIGAN).—In reply to Dr. Singer's statement that one cannot divide the pulmonary abscess cases into surgical and medical, I thought I made that point emphatic in the reading of my paper. Those cases that do not improve on medical treatment, of course, should have surgical drainage. But when the abscess ruptures into the bronchus and drains thoroughly with postural treatment, I do not see why we should not let them recover in that way rather than perform an operation on them and maybe have them die.

Dr. Cole says, "Let them alone when they are acute." I say so, too. But if the abscess fails to drain, then, of course, surgical treatment is indicated. I think we would have far better results in the treatment of lung abscess if we let more of them alone.

THE MEDICAL TREATMENT OF ABSCESS OF THE LUNG*

CONRAD GEORG, JR., M.D.
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ALL patients with lung abscess should receive medical treatment during the first two months of its course. About 30 per cent of the patients recover under a medical regimen, provided the abscess ruptures into a bronchus and drains freely. If healing does not progress satisfactorily and there is evidence of retention of secretions accompanied by chills and fever, with foul sputum and progressive loss of flesh and strength, one should not delay bronchoscopic and surgical treatment, as the danger to life from a continuance of these conditions is greater than the risk of operation in carefully selected cases. Multiple abscesses of the lung, however are not amenable to surgical treatment, the mortality being 100 per cent. A case of this kind was reported at a previous meeting of this society. There are also some cases in which adequate drainage is impossible by surgery and others in which the system is overwhelmed with toxins and operations only hasten the end and are a discredit to good surgery.

Under the etiology of abscess of the lung may be mentioned aspiration of pyogenic organisms from diseased teeth due to pyorrhea or septic tonsils, especially after tonsillectomies performed under general anesthesia. Septic emboli may infect the lung from the blood stream as in a case previously reported or infection may extend from neighboring tissues. When the inflammation becomes circumscribed, suppuration takes place in the interior of the focus resulting in the formation of a cavity. Davies[4] says that there is an area of pneumonic consolidation around a suppurating focus in the lobe of a lung and that there is no sharp line of demarcation between the diseased and healthy tissue. The parenchyma of the lung breaks down with a large or small amount of débris. In the former case there may be considerable elastic tissue in the sputum. Spector[3] found in 35 cases that elastic tissue was not always present. In fact its presence is not diagnostic of lung abscess because it is present in cases of ulcerative inflammation of the trachea and larynx. The abscess sometimes ruptures into a bronchus with the escape of pus into the sputum. Abscesses located near the external surface of the lung may rupture into the pleural cavity with the formation of an empyema. Drainage through a bronchus may result in a cure, especially if the abscess is in the upper lobe. An abscess may drain well without effecting

*Read at the Fifteenth Annual Meeting of the American Association for Thoracic Surgery at Ann Arbor, Michigan, April 18, 19, 20, 1932.

a cure when it becomes converted into a chronic abscess with the formation of a wall of fibrous tissue around it.

An abscess which has continued more than two months should be regarded as chronic. In gangrenous cases the breath becomes very foul, the pus has a bad odor and is dark in color from broken down blood, and it contains elastic fibers.

About 70 per cent of the cases of lung abscess follow surgical operations. Of this group about 56 per cent follow tonsillectomies. Acute infections of the respiratory tract, including pneumonia and bronchopneumonia cause about 25 per cent of the cases, while malignant disease accounts for about 10 per cent of the cases.[5] Whittemore[6] states that of 100 cases, 66 followed operations upon the upper respiratory tract under a general anesthetic. Of these, 48 were tonsillectomies and 12 extraction of teeth. Bronchopneumonia was the cause in 20 of them. Lilienthal[7] found that tonsillectomy was the cause in 21 of 105 cases and pneumonia including influenza in 29. Schlueter and Weidlein[8] found that in 1908 cases, 26.9 per cent followed operations and 14.6 per cent followed tonsillectomies. The majority of these cases follow operations under general anesthesia. Moore[9] stated that 159 cases out of 202 followed tonsillectomies done under general anesthesia while 39 cases followed the same operations done under local anesthesia. It comes on between the tenth and fifteenth days after tonsillectomy.

Hedblom discussing 692 cases of lung abscess at the Mayo Clinic, says, "the great majority of pulmonary complications following tonsillectomy are due to aspiration infection, and the great majority of them followed operation under general anesthesia."

Davies[4] considers that it is more than probable that both aspiration and embolism play an important part in the causation of lung abscess and that in each case the development of the abscess is dependent upon a combination of both trauma and sepsis. The aspirated matter acts as a trauma by either causing a local inflammatory reaction or occluding a bronchiole and on the other hand the embolus produces interference with the blood supply.

The organisms which are concerned in the causation of lung abscess are the Staphylococcus aureus, the streptococcus, Bacillus coli, spirochetes, and the anaerobic Bacterium melaninogenicum. Crowe and Scarff[10] say, "The spirochetes in the mouth have been regarded as secondary invaders, but it is possible that they may play an important rôle in the etiology of abscess of the lung which follows operations upon tonsils and teeth." It has also been demonstrated that the spirochetes are present in cases of pyorrhea alveolaris and in pus expressed from crypts in the tonsils and are believed to be the cause of the offensive odor in chronic lung abscess. Burdon[16] and Varney found that the Bacterium melaninogenicum produces a black pigment on suitable culture

media and in symbiosis with other organisms produces extensive necrosis of tissue and the cultures develop a foul odor similar to that found in lung abscess.

Davies[4] says that the right lobe is involved three times as often as the left and the lower lobe twice as often as the upper and the ratio of men to women who are afflicted with it is as three to one.

SYMPTOMS AND SIGNS

During the acute stage there is usually violent coughing, chills and fever of 104° or 105° F. The sputum at first is small in amount and streaked with blood and the symptoms are similar to those of beginning pneumonia. Dyspnea is not always present. There is usually a rapid loss in weight and strength. In about ten or fifteen days the abscess usually ruptures into a bronchus when large quantities of foul sputum are expectorated, as much as 500 c.c. or more in twenty-four hours. There is often an offensive breath even though there is no sputum. The latter is usually of a mucoid character before the abscess ruptures. The sputum may be colored with bright blood or it may be brown in color from broken down hemoglobin. There are often elastic fibers in the sputum. Three layers will be noticed in the sputum after it has stood for a few days. The upper layer consists of frothy mucus, the middle layer is a turbid liquid and consists mostly of saliva and the bottom layer consists of pus. A foul odor is usually present in a case of gangrene.

Failure to expectorate the sputum may be accompanied by a rise in temperature. This condition may be followed by the discharge of large quantities of bloody, foul-smelling sputum. The signs of a deep-seated abscess may be absent, although pronounced symptoms of such a condition may be present. Dullness upon percussion may be noticed when there is a superficial abscess. The breath sounds are diminished when auscultation is practiced. In the case of a large cavity the percussion note becomes tympanitic and bronchial breathing will be heard over it. There also will be moist, coarse and fine râles.

X-ray examination should always be made to determine the extent of involvement of the lungs and whether there are single or multiple abscesses. The location of the abscess usually appears as a dense shadow, surrounded by an area of consolidated lung tissue which gives a less dense shadow. When a cavity containing pus is formed, a fluid level can be seen with a rarefied area above.

TREATMENT

Our treatment for cases of abscess of the lung has been rest for a period of at least two months during the initial stage of its course. More or less continuous postural treatment was adopted. The length of time for the continuance of this treatment depends upon the strength of the

patient. When treated in this way the pus continues to pour out in large quantities until the drainage is complete. In the course of a month or six weeks the amount of expectoration becomes reduced to about one tablespoonful of glairy mucus in twenty-four hours. Surgical treatment must be employed when the abscess fails to rupture into a bronchus. Bacteriophage was used in addition to the above medical regimen. Twort[12] says that bacteriophage is a bacteriolytic filtrable and transmissible agent which was described by him previous to the work of d'Herelle. It is constantly found in polluted water. F. d'Herelle was the first to point out the value of bacteriophage in the treatment of acute septic infections. Our bacteriophage was furnished by the Michigan State Board of Health at Lansing.

D'Herelle believes that bacteriophage is a living ultramicroscopic corpuscle which dissolves bacteria through the agency of a ferment which it secretes. The latter implies that it is a process of metabolism. Other bacteriologists do not agree with d'Herelle that bacteriophage is a living corpuscle, but consider it to be a chemical ferment, the product of bacterial action. It may dissolve bacteria belonging to different species or it may only attack a single species or strain. Patients affected with bacillary dysentery were given 2 c.c. of a culture of bacteriophage, and in all cases the patients improved within a few hours. In 1927 d'Herelle treated Asiatic cholera in India with a mortality of 8.1 per cent, while the mortality among the controls was 62.9 per cent. It has also been used in cases of streptococcus septicemia with good results. In cases of pulmonary abscess I have given a series of ten to fifteen doses of 2 c.c. intramuscularly with good results. D'Herelle says that suspensions of bacteriophage are perfectly clear and the slightest turbidity indicates contamination; such material should not be used.

Patients with lung abscess should be supplied with highly nourishing food often. Our internal medication consists of terpin hydrate, silicic acid, and calcium glycerophosphate.

SUMMARY

All patients with lung abscess should receive medical treatment during the beginning of their course. Good results were obtained in several single lung abscesses and in some chronic abscesses. Patients who do not improve under medical treatment within two months should be subjected to bronchoscopic or surgical treatment. Bacteriophage was found to be of value in the medical treatment of lung abscesses.

REFERENCES

1. Georg, Conrad, Jr.: Abscess of the Lung Following Fracture of the Rib, Arch. Surg. **18**: 526, Part II, 1929.
2. Flick, J. B., Clerf, L. H., Funk, E. H., and Farrell, J. T., Jr.: Pulmonary Abscess, Arch. Surg. **19**: 1292, Part II, 1929.
3. Spector, H. I.: Lung Abscesses, J. A. M. A. **95**: 789, 1930.

4. Davies, H. Morriston: Surgery of the Lung and Pleura, 140-159, 1930.
5. Graham and Wiese: Arch. Surg. **16**: 380, 1928.
6. Whittemore: Surg. Gynec. Obst. **38**: 461, 1924.
7. Lilienthal: Arch. Surg. **16**: 206, 1928.
8. Schlueter and Weidlein: Arch. Surg. **14**: 457, 1927.
9. Moore: J. A. M. A. **78**: 1279, 1922.
10. Crowe, S. J., and Searff, J. E.: Experimental Abscess of the Lung in the Dog, Arch. Surg. **16**: 176, 1928.
11. D'Herelle, F.: Bacteriophage as a Treatment in Acute Medical and Surgical Infections, Bull. New York Acad. Med. **7**: 329-348, 1931.
12. Twort, F. W.: Filtrable and Transmissible Bacteriolytic Agents (Bacteriophage), Ann. Inst. Pasteur **47**: 459-578, 1931.
13. Graham, E. A.: Surgical Diagnosis, Vol. III, pp. 56-77.
14. Singer, J. J., and Graham, E. A.: A Study of 34 Cases of Abscess of the Lung, J. A. M. A. **81**: 193, 1923.
15. Ermatinger, Lucille H.: Microorganisms of Lung Abscess and Bronchiectasis, J. Infect. Dis. **43**: 391-398, 1928.
16. Burdon, Kenneth L.: Bacterium Melaninogenicum From Normal and Pathologic Tissues, J. Infect. Dis. **42**: 161-171, 1928.
17. Allen, Duff: Etiology of Abscess of the Lung, Arch. Surg. **16**: 179, 1928.
18. Smith, David T.: Experimental Aspiratory Abscess, Arch. Surg. **14**: 231, 1927.
19. Lemon, Willis S.: Aspiration, An Experimental Study, Arch. Surg. **12**: 187, 1926.
20. Iglauer, S.: Ann. Otol. Rhin. & Laryng. **37**: 231, 1928.

DISCUSSION

The discussion of this paper will be found with that of Dr. Neuhof's paper at the end of Dr. Neuhof's paper, p. 646.

THE THORACIC COMPLICATIONS OF SUBDIAPHRAGMATIC INFECTION*

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INTRODUCTION

INFECTIONS in contact with the under surface of the diaphragm may be divided into those which are contained within the peritoneal cavity and those which lie behind the peritoneum.

The *intraperitoneal group* is the largest and most important and is generally considered when the term subdiaphragmatic or subphrenic abscess is used. Etiologically such infections have their inception for the most part (75 per cent to 80 per cent) in perforation of a peptic ulcer, duodenal or gastric, or in general contamination of the peritoneal cavity extending from an acutely inflamed appendix. The infecting organism is commonly a streptococcus or the B. coli alone or in combination. Lying between the upper surface of the liver and the diaphragm, the infection tends to become promptly walled off. The diaphragm forms the roof of the abscess.

In the *extraperitoneal group* the infection is usually perinephritic in character and the infecting organism is the Staphylococcus aureus in about 75 per cent of all cases. However, in only a small number of all cases of perinephritic abscess does the infection invade the fatty capsule above the upper pole of the kidney to make contact with the diaphragm. Occasionally an acutely inflamed appendix which lies in the retroperitoneal position may be responsible for an infection which extends upwards behind the peritoneum to reach the diaphragm. The infecting organism is then likely to be a streptococcus or B. coli.

Any infection in contact with the diaphragm is inherently likely to extend through the latter to produce a supraphrenic inflammation. The greater the surface of the diaphragm involved in the subphrenic infection the more likely is such extension to occur. In the intraperitoneal variety, therefore, transphrenic infection is relatively much more common. In this group, furthermore, the frequent presence of the streptococcus alone or in mixed infection is an important factor.

EXTENSION OF INFECTION THROUGH THE DIAPHRAGM

The diaphragm is traversed by a rich network of lymphatic vessels which drain into a series of nodes lying upon its superior surface. The lymph flow is from below the diaphragm into these nodes. The latter

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are covered by the pleura which lines the upper surface of the diaphragm.

When an inflammation develops acutely and suddenly under the diaphragm and especially if a wide area of the latter is involved, the inflammation may extend rapidly *via the lymphatics* to produce a reaction of the pleura. This may be represented by a *pleural effusion* which early is serous, sterile and of moderate amount. The presence of exudate in the pleural cavity is readily detected by physical findings and roentgenologic study.

An early pleural effusion is not uncommon following the acute perforation of a peptic ulcer and may be one of the earliest signs of the development of a subphrenic abscess. Developing during the postoperative course after suture of a perforation, the effusion may be mistaken for a pneumonic process or a massive collapse (atelectasis) of the lung.

Occasionally the development of an effusion within the pleura will be both abrupt and in very large amount.

While the early serous pleural effusion is usually protective in nature, such as is commonly seen in any serous lined cavity in contact with an inflammatory process, it may contain organisms. Such bacteria will sometimes be seen in stained smears of the exudate but fail to grow on media. In other cases the bacteria may be grown actively on culture media.

A serous pleural exudate is not always an early manifestation of a subphrenic infection. It may develop only after the latter has existed for weeks or months. Furthermore, an effusion may exist as only part of the thoracic pathology. There may be an associated pneumonitis or walled off empyema.

The course of the pleural effusion is significant. With the establishment of drainage of the subphrenic infection, the pleural exudate may be rapidly and completely absorbed even if it contains viable bacteria. Increasing evidence of infection of the serous effusion is not uncommon until it is frankly purulent (empyema) and this may occur even though the subphrenic infection is adequately drained by a surgical attack.

The earliest manifestation of a supraphrenic inflammation is commonly a *plastic pleuritis* which fixes the under surface of the lower lobe of the lung to the diaphragm. This is often indicated clinically by "pleuritic pain" on breathing, sometimes refered to the root of the neck on the affected side, and when studied by roentgenograms the surface of the diaphragm is indistinct, "fuzzy," in outline.

If gross infection develops above the diaphragm in a case in which the initial reaction has been an obliterative pleuritis, the pathologic course is variable. As pus forms it may remain sharply localized between lung and diaphragm, limited in amount. The amount of pus may increase more or less gradually and strip the adhering visceral and diaphragmatic

pleural surfaces apart to reach the thoracic wall. Such an empyema is frequently loculated, with bands of fibrinous or fibrous (depending upon its duration) adhesions passing from the pulmonary to the diaphragmatic pleural surfaces.

An empyema may develop subsequent to adequate drainage of a subphrenic infection. This is due to the fact that bacteria have traversed the diaphragm and are lodged in fibrin between the diaphragm and lung. Following drainage and release of tension the diaphragm tends to recede to a lower level and strip away from the adherent lung, thus lighting up the infection within the pleura.

An empyema developing in a plastic pleural exudate will often invade the lung parenchyma as a course of least resistance, to establish communication with a bronchus.

Pneumonitis in some degree is frequently seen. It may be represented as a limited area of consolidation of that portion of the lung adjacent and adherent to the diaphragm. This may be an inflammation without infection and tends to subside promptly if drainage of the subphrenic infection is adequately established. In other instances actual invasion of the lung parenchyma occurs with the development of one or more areas of pulmonary suppuration. Extension of an empyema into lung substance has already been mentioned.

In any case, the pathologic process may be a complicated one. One or more empyemata and pneumonitis may be present as independent processes. There may be pneumonitis and an independent serous effusion, or an empyema and serous effusion.

In certain cases the subphrenic infection may have established or maintained communication with a hollow viscus and when the infection traverses the diaphragm the picture becomes bizarre. Fistulous communication may develop between some portion of the gastrointestinal tract and the bronchial tree.

Extension through the diaphragm in the earliest stages of the subphrenic infection is usually via the lymphatics. As the subphrenic process becomes firmly localized to form an abscess, there is an inherent tendency for the diaphragm to become grossly invaded. This frequently terminates in a *perforation of the diaphragm* and the establishment of a direct communication between the subphrenic infection and the thoracic pathology.

The site of perforation is variable and depends upon the level of "pointing" of the subphrenic involvement. In an occasional case there will be more than one perforation. There may be several, suggesting that necrosis of the diaphragm took place over a considerable area preceding the gross breaks.

The size of the perforation through the diaphragm varies from that of a probe to several centimeters in diameter. In most instances the defect is of such size as readily to allow the insertion of one's index finger.

The development of the perforation is usually gradual and is preceded by the development above the diaphragm of a plastic pleuritis, empyema or pneumonitis. Occasionally perforation takes place into an area of pleural space harboring a serous exudate, and the latter is converted abruptly into an empyema.

If an empyema has been present before perforation of the diaphragm occurs or develops subsequently, a *pyopneumothorax* may result if the subphrenic abscess has contained air. This is seen in cases where the subphrenic infection has followed the perforation of a hollow viscus. Similarly a lung abscess which has formed no connection with a bronchus may contain air.

The pus contained within an empyema is of the same character as that of the subphrenic abscess unless the former has established drainage into the bronchial tree and has become secondarily infected. If the infecting organism is the B. coli or B. proteus, the pus will be foul smelling.

CLINICAL SIGNIFICANCE OF THE THORACIC COMPLICATION

The importance of the thoracic complication is in relation especially to diagnosis and therapy.

Diagnosis.—When a subphrenic infection is known to exist it is not difficult to recognize the development of an extension of infection above the diaphragm. The first diagnostic step is to bear in mind the possibility or likelihood of such extension.

Often in an acute subphrenic infection such as may follow perforation of a peptic ulcer several days must elapse before localization takes place beneath the diaphragm. During this period there is a general and leucocytic reaction which is readily explainable on the known infection. A transphrenic extension may develop without any change in the general reaction.

Daily careful examination of the chest must be made to discover evidence of significant change within the thorax. Roentgenologic study will disclose the findings of pleuritis or pneumonitis. If fluid accumulates it is wise to perform thoracentesis early to determine its character.

One should bear in mind that a supraphrenic infection may manifest itself only after a subphrenic abscess has been drained. In such a case one might be inclined to explain a continuation of fever, rapid pulse, or increased leucocytosis on the basis of inadequate drainage of the subphrenic involvement and fail to recognize the thoracic complication.

It is not uncommon that the findings of the thoracic involvement give the first clue to the presence of an acute subphrenic abscess in a patient who is suffering from an unlocated abdominal infection. This is particularly true in cases in which a diffuse peritoneal contamination has followed an attack of acute appendicitis and in small perinephritic abscesses around the upper pole of the kidney.

It is in unsuspected subphrenic abscess that the thoracic complications offer the greatest difficulty in proper interpretation. A subphrenic abscess may develop insidiously and without recognizable cause. It may be almost unbelievably chronic and lay dormant for months or years. Thoracic complications from a hidden or chronic subphrenic abscess may develop suddenly, or insidiously at any time during the course of the chronic and unsuspected abscess and produce a clinical picture which is confusing. Such a patient is often treated for pulmonary tuberculosis, bronchiectasis or primary empyema. Surgical drainage of such an empyema is likely to be followed by a chronic rest cavity or recurrence of the empyema.

In this group a very carefully taken history will often give important diagnostic clues, leading to the recognition of the subphrenic infection. Prolonged convalescence following an attack of appendicitis with or without operation; the history of pelvic infection complicating childbirth; an unusually severe and prolonged attack of "stomach trouble" are often very significant.

Subphrenic abscess is seldom stumbled upon by diagnostic accident. The possibility of its presence must be considered in any case of confusing intrathoracic infection. An empyema containing a foul smelling pus should arouse suspicion of a transphrenic infection.

Sometimes the presence of a subphrenic abscess is only discovered when an empyema is drained and the gross communication through diaphragm is found by palpation.

TREATMENT

When the supraphrenic reaction is not suppurative, it may subside promptly and completely with adequate drainage of the subphrenic infection. Large serous pleural effusion will often be absorbed within a few days after subphrenic drainage. All evidence of pneumonitis may disappear, leaving a clean cut diaphragm outline as seen in chest films.

When there is a gross suppuration both above and below the diaphragm adequate treatment will demand drainage of each independently as a general rule. This is especially true when the supraphrenic infection is an empyema.

In any case, drainage of only one of the purulent accumulations may determine a cure of the other if there happens to be present a gross defect in the diaphragm. It is not uncommon for a suppurative pneumonitis with or without established communication with the bronchial tree to subside completely following drainage of the subphrenic abscess.

TABLE I.—ETIOLOGY OF SUBPHRENIC INFECTION, INTRAPERITONEAL. 31 CASES.

With thoracic complications—23:		Without thoracic complications—8.
Peptic ulcer	10	4
Appendicitis	10	2
Pelvic infection	1	1
Liver abscess	1	0
Infected dermoid cyst	1	1

TABLE II.—Bacteriology of Subphrenic Infection, Intraperitoneal. 31 cases.

	With thoracic complications—23:	Without thoracic complications—8.
Mixed, including a streptococcus	9	5
Streptococcus alone	1	2
Colon	4	1
Not recorded	9	

TABLE III.—Type of Thoracic Complication: Subphrenic Infection, Intraperitoneal. 23 cases.

Empyema	10
Empyema with bronchial communication	3
Pleural effusion, simple	1
Pleural effusion, infected	1
Pneumonitis, mild	3
Pneumonitis, suppurative	2
Pneumonitis with bronchial communication	3

GROSS PERFORATION OF DIAPHRAGM—15 cases, or 65.2 per cent.

TABLE IV.—Gastrointestinal Communication With Thoracic Pathology. 4 cases.

Hepatic flexure of colon	1
Cecum	1
Appendix	1
Transverse colon (Broncho-colic fistula)	1

TABLE V.—Side Affected by Thoracic Complication. 23 cases.

Right	17
Left	6

TABLE VI.—Onset of Thoracic Complication, Subphrenic Infection, Intraperitoneal. 23 cases.

Abrupt	2
Early	12
Gradual	3
Late	6

Two patients were treated for pulmonary tuberculosis.

In *six* patients the thoracic complication led to the diagnosis of subphrenic infection.

TABLE VII—Mortality, Subphrenic Infection: Intraperitoneal.

With Thoracic Complications	23 cases:	10 deaths—43.5%
Without Thoracic Complications	8 cases:	2 deaths—25%

TABLE VIII.—Treatment. Subphrenic Infection, Intraperitoneal, With Thoracic Complications.

Drainage of subphrenic infection and empyema independently
8 cases: 7 recoveries 1 death

Drainage of subphrenic infection, empyema not drained
3 cases: No recoveries 3 deaths

Drainage of empyema, subphrenic infection not drained
2 cases: 1 recovery 1 death

Drainage of subphrenic infection, pneumonitis not treated
6 cases: 3 recoveries 3 deaths

Drainage of subphrenic infection, infected effusion not treated
1 case with recovery

Drainage of subphrenic infection, simple effusion present
1 case, death

Not treated: 2 cases
1 Spontaneous drainage of subphrenic infection through bronchus—recovery
1 Early death from subphrenic infection with pneumonitis

TABLE IX.—Bacteriology of Subphrenic Infection, Perinephritic. 25 cases.

	With Thoracic Complications—8:	Without Thoracic Complications—17.
Staphylococcus	6	12
Streptococcus	1	0
Colon	0	1
Mixed	0	1
Not recorded	1	3

TABLE X.—Thoracic Complications. Subphrenic Infection, Perinephritic. 8 cases.

Empyema	3
Infected Effusion	2
Simple Effusion	2
Pneumonitis	1

PERFORATION OF DIAPHRAGM—2 cases—25%.

TABLE XI.—Mortality. Subphrenic Infection, Perinephritic. 25 cases.

With Thoracic Complications	8 cases:	2 deaths—25%
Without Thoracic Complication	17 cases:	1 death—5.8%

SUMMARY

1. In 31 cases of intraperitoneal subphrenic infection thoracic complication occurred in 23, or approximately 75 per cent.

2. In 23 cases of intraperitoneal subphrenic infection with thoracic complication there was gross perforation of the diaphragm in 15, or approximately 65 per cent.

3. In 25 cases of perinephritic subphrenic infection thoracic complications occurred in 8, or 32 per cent.

4. In 8 cases of perinephritic subphrenic infection with thoracic complication there was gross perforation of the diaphragm in 2, or 25 per cent.

5. Empyema is the commonest thoracic complication of subphrenic infection.

6. A subphrenic infection produced by the streptococcus in combination with the B. coli or B. proteus is especially likely to traverse the diaphragm.

7. A thoracic infection may develop acutely or insidiously as a complication of subphrenic infection.

8. The thoracic complication may be the first diagnostic clue to the presence of a subphrenic infection.

9. When empyema develops as a complication of subphrenic infection, drainage must be established of both areas independently, as a general rule.

10. The mortality of subphrenic abscess is much higher when there is a thoracic complication.

11. There is an inherent tendency for an infection in contact with the under surface of the diaphragm to traverse the latter to produce a thoracic infection because of the course of the lymph flow.

12. The diaphragm is a very efficient barrier to extension of infection from the thorax to the abdomen. In a series of 337 cases of empyema

to January 1, 1932, in the University Hospital there has been only one instance in which infection traversed the diaphragm to produce a subphrenic abscess and that was due to injury of the diaphragm during drainage of an empyema.

DISCUSSION

DR. FREDERICK T. LORD (BOSTON).—Elevation of the diaphragm from the accumulation of pus below it gives physical signs which differ according to the degree of encroachment on the lung. With small subdiaphragmatic abscesses dullness and diminished breathing may be the only significant changes, but it is important to appreciate that a large purulent accumulation below the diaphragm may readily be confused with empyema. There may then be an extensive area of dullness posteriorly, with diminished and bronchial breathing, increased whisper, egophony and absent tactile fremitus. The heart is likely to be less displaced than with an equal amount of pleural fluid and relative resonance may be noted in the paravertebral region on the side corresponding to the subdiaphragmatic abscess. Roentgen-ray examination is desirable in all such cases to avoid confusion with empyema and the production of open pneumothorax by operation.

DR. HOWARD BEYE.—I made no attempt to discuss the treatment of these cases thoroughly, because of the limited time at my disposal. I felt that the pathologic considerations were of greater importance.

One point I did not make that I want to make now, in closing, is that in 337 cases of empyema which we have had, chronic and acute, there was only a single instance of infection above the diaphragm traversing the diaphragm to produce an infection of the abdominal cavity. That was a case in which, in draining the empyema, we traumatized the diaphragm. I think it is significant that there is an inherent tendency for the diaphragm to be an efficient barrier to the transference of the infection from above it to below it, but the contrary is in our experience the rule rather than the exception.

ANTERIOR MEDIASTINAL FETAL PARASITE; ITS SURGICAL REMOVAL: REPORT OF A CASE*

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TERATOID growths may be found in many parts of the body. They are most commonly situated, in order of frequency, in the pelvic, abdominal, sacrococcygeal and thoracic regions. The relative infrequency of the incidence of these tumors in the thorax, and the rarity of fetal tissue in growths which present a sufficiently high degree of organoid structures to be designated as a fetal parasite are of sufficient interest to warrent the following report.

The patient, a girl aged seventeen years, had first noticed slight pain in the left anterior part of the thorax four years before her admission to the clinic. The pain had occurred at irregular intervals and was more in the nature of soreness than pain and was usually noted when she contracted a severe cold, more particularly associated with bronchitis and coughing. One and a half years before, she first had noted swelling in the lower left side of the neck just posterior to the sternal end of the clavicle which extended into the suprasternal notch. The condition was believed to be due to enlargement of cervical glands and was treated by local application of ointment, following which the mass disappeared completely. About a year later severe bronchitis associated with coughing developed, and the tumor reappeared. Local treatment was again instituted, but the tumor did not disappear and operation was advised. It was performed about two months later, and the patient stated that a large amount of sebaceous material containing hair was removed from the anterior mediastinum but the tumor was not removed. After the operation, the patient had some pain in the upper part of the back between the shoulder blades, and particularly in the back of the neck. About two weeks before her admission the tumor again appeared in the neck beneath the operative incision in the skin and rapidly increased in size. Its situation had shifted more toward the median line in the suprasternal notch. At the time of her admission, there was a great deal of drainage of infected sebaceous material from the wound, and a large mass projected beneath the skin extending into the anterior mediastinum. There had been a loss of ten pounds in weight in the last three months, which was attributed to her present condition.

The blood pressure in millimeters of mercury was 105 systolic, and 68 diastolic. The pulse rate was 120 beats each minute. The temperature was of a moderately septic type ranging from 99.2 to normal. A tumor in the supersternal notch just above the sternal end of the clavicle, as described, was found. The concentration of hemoglobin was 13.7 gm. for each 100 c.c.; erythrocytes numbered 4,690,000 and leucocytes 8,600 in each cubic millimeter of blood. Urinalysis was negative. The Wassermann reaction of the blood was negative. The roentgenologic examinations of the thorax consisted of anteroposterior stereoscopic views, lateral views, and fluoroscopy. These examinations revealed a tumor without pulsation in the upper

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part of the mediastinum extending into both thoracic cavities. The tumor was more in the anterior part of the mediastinum than in the posterior. The roentgenologists were unable to separate the shadow of this tumor from that of the arch of the aorta (Fig. 1). A clinical diagnosis of anterior mediastinal tumor, probably teratoid, was made and operation advised.

January 31, 1931, anterior mediastinotomy was performed. Intratracheal gas anesthesia was used because of the possibility of injury to either or both mediastinal parietal pleurae which if it had occurred would result in unilateral or bilateral pneumothorax. The anterior method of approach to the tumor was made by excising and enlarging the incision at the base of the neck, which was made at the previous operation done elsewhere, and it was necessary further to enlarge the open-

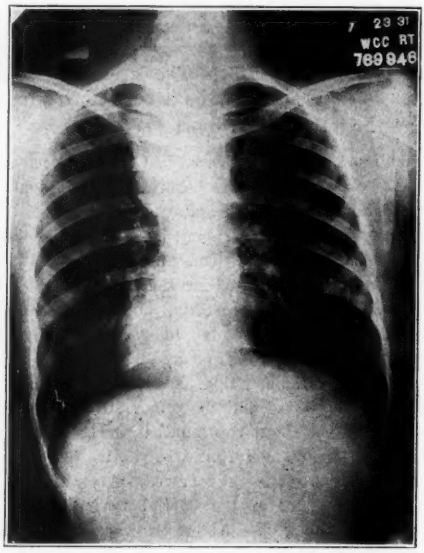


Fig. 1.—Tumor in the upper part of the mediastinum projecting into both thoracic cavities, more marked on the left. The shadow could not be separated from the arch of the aorta. Fluoroscopic examination showed the tumor to be nonpulsile and in the anterior mediastinum.

ing by partially splitting the sternum in order to obtain sufficient exposure to remove the tumor. This approach was made to avoid, if possible, contamination of one or both pleural cavities which may have resulted from the infected material around the tumor if another type of approach such as a transpleural operation had been attempted. The tumor extended deep into the anterior mediastinum under the arch of the aorta, protruded into the left thoracic cavity at the level of the sixth rib, and extended into the right thoracic cavity at a slightly higher level. There were several budlike processes extending from the main tumor, and its lower portion extended beneath the arch of the aorta to which it was adherent. The tumor was attached to a moderate extent to the posterior surface of the sternum by fibrous bands. Considerable bleeding was encountered in removing the growth from its attachment to the aorta and it was necessary to ligate several moderate sized vessels which entered the growth at its lower pole; these vessels were undoubtedly the source of its blood supply. The entire tumor was removed and the sebaceous

material was sponged out of the large cavity which was found to be lined with a friable serous membrane firmly adherent to the adjacent pleura; as much as possible of this membrane was removed; it seemed to be absent at the lower part of the cavity around the arch of the aorta, or it may have been so firmly adherent to the surrounding structures that it was not discernible. The large anterior mediastinal cavity was packed with gauze and the wound was partially closed (Fig. 2).

There was a moderately severe reaction during the operation, and the patient was given intravenous injections of gum acacia and saline before leaving the operating table. She was placed in an oxygen cabinet after leaving the operating room. The tumor weighed 132 gm. and measured 8 by 7 by 5 cm. Grossly the tumor was

Fig. 2.—Anterior mediastinal growth. Its irregular spheroid shape with four definite budlike projections covered with normal-appearing piliferous skin are shown. The tumor measured 8 by 5 cm. and weighed 132 gm.

a large irregular spheroid-shaped mass from which there were four budlike processes. The entire tumor was covered with thick, normal-appearing piliferous skin.

The pathologic diagnosis following microscopic examination was as follows: "The tumor represents the caudal portion of a parasitic fetus covered with skin and containing: (1) dermal structures, such as, hair and hair follicles, sebaceous and sweat glands, fatty connective tissue, lymphoid tissue and nerve tissue; (2) several cavities lined by columnar epithelium with goblet cells. There are also in the walls of these cavities mucous glands which are suggestive of the lower part of the intestinal tract; (3) masses of ossifying cartilage containing marrow, surrounded by dense fibrous and ligamentous and voluntary muscle tissue, structures

which probably represent the lower portion of the spine; and also one small island of cartilage situated in the region which may be of the symphysis pubis; (4) smooth muscle coat encircling the wall of a large cavity which is lined with transitional and columnar epithelium; this structure is suggestive of a cloaca, and (5) a large circumscribed mass of pancreatic tissue containing one large duct and many islands of Langerhans'' (Figs. 3 to 8).

A moderate reaction followed the operation; convalescence was satisfactory. The pulse rate was 130 and temperature 101° F. on the second day and saline was given intravenously. The temperature and pulse rate fell to normal on the fifth day, remaining practically normal thereafter. The patient was dismissed from hospital on the eighteenth day and from observation on the thirty-seventh day after operation

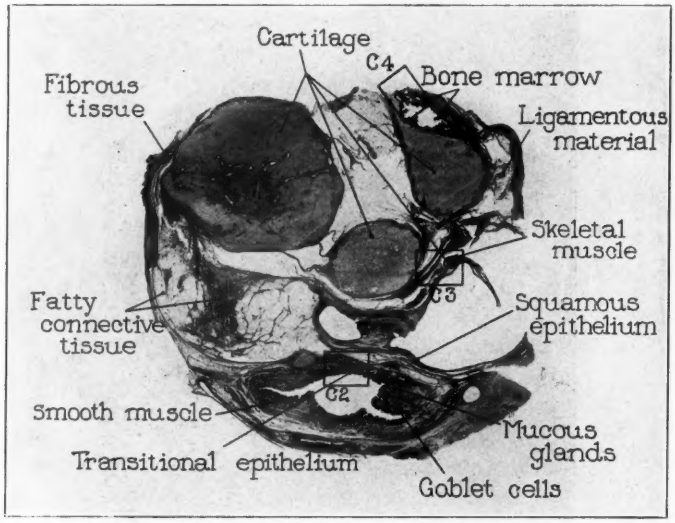


Fig. 3.—Cross section of parasitic fetus, probably lumbar region.

at which time the wound was practically healed except for a small granulating sinus. She had gained eighteen pounds in weight since the operation and her general condition was very good. In reply to a letter of inquiry one year after the operation, she stated that her general condition was very good and the wound was entirely healed. She had had no further symptoms (Fig. 9).

My experience in the treatment of intrathoracic tumors comprises a series of thirty-six, all of which were removed by transpleural operation except the case here reported. Of this group, eight were of teratoid type of varying degrees of complexity. Six were fetal tissues containing growths, one was a squamous-cell epithelioma, evidently malignant degeneration of a teratoid tumor, and the case reported here presented a sufficiently high degree of organization to be termed a fetal parasite.

This case is of clinical interest because of the apparent long duration, large size, and situation of the tumor, and the resulting mild symptoms.

The chief surgical considerations and points of interest were the difficulty of access through the anterior approach and the necessity of splitting the sternum before the tumor could be delivered from the anterior mediastinal pocket. It was necessary to avoid injury of the mediastinal pleurae because of the danger of producing empyema, since the pocket of infected sebaceous material and hair surrounding the tumor was encased in a thin friable membrane which was firmly adherent to the pleura in many places. When adequate exposure was obtained by splitting the sternum, this serous membrane was carefully dissected

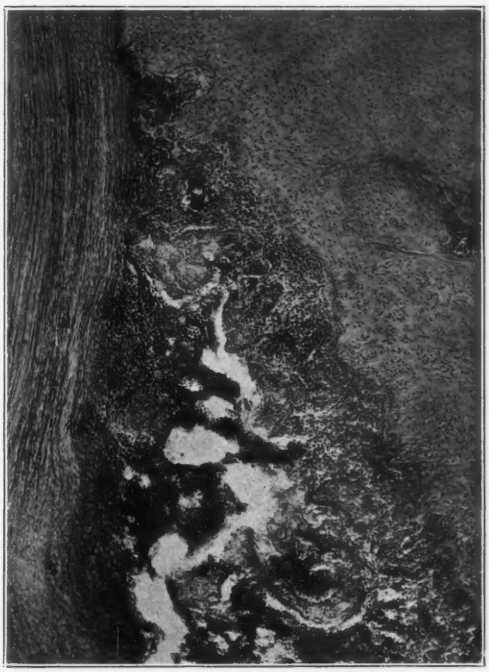


Fig. 4.—Area *C4* (Fig. 3) of lumbar cross section of parasitic fetus; ossifying cartilage and bone marrow formation are shown.

away from the pleura from above down, but in some places it was so adherent that small areas were left attached. The difficulty encountered in separating the deep attachment of the growth which extended beneath the arch of the aorta where bleeding was encountered, necessitated careful hemostasis. The chief interest, however, is centered in the gross and histologic appearance of the tumor and the rarity with which a tumor of this type is found entirely within the thoracic cavity. Grossly the tumor suggested an unusual type, possibly of fetal origin, because

of the rounded oblong shape of the main part of the growth which was entirely covered with normal-appearing thick piliferous skin with four budlike outgrowths. The histologic appearance of the growth was of

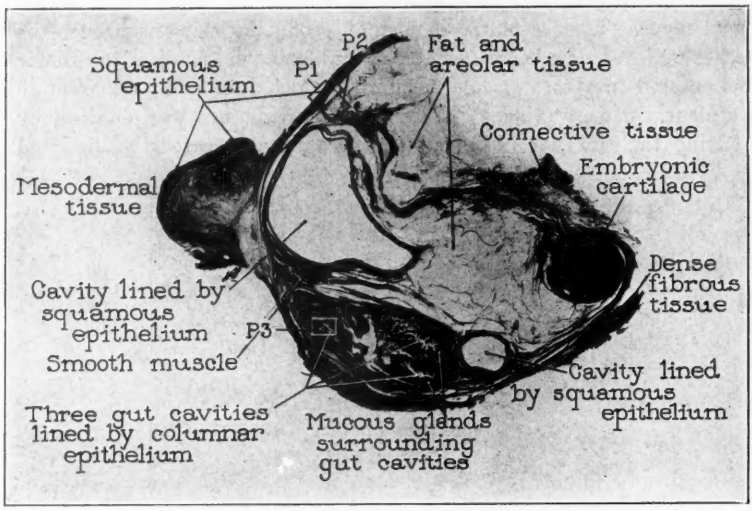
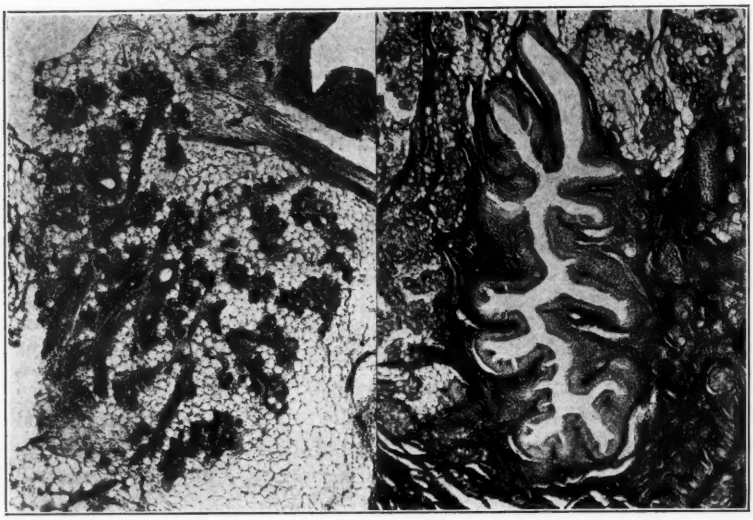


Fig. 5.—Cross section of parasitic fetus through one of the posterior budlike projections.



A *B*

Fig. 6 *A*.—Section PII (Fig. 5), from posterior portion of parasitic fetus showing compound racemose glands lying in subcutaneous fat suggesting developing mammary gland; *B* section PIII (Fig. 5) from posterior portion of parasitic fetus; one of three gut cavities with mucous glands in its wall are shown.

greatest significance as it revealed embryonic and adult tissues derived from all three layers of the blastoderm. The arrangement of these tissues, as shown by serial cross sections, disclosed a marked degree of organoid structure. I have been unable to find in the literature any other case in which the tumor contained so much organoid structure both grossly and histologically, that was entirely within the thoracic cavity

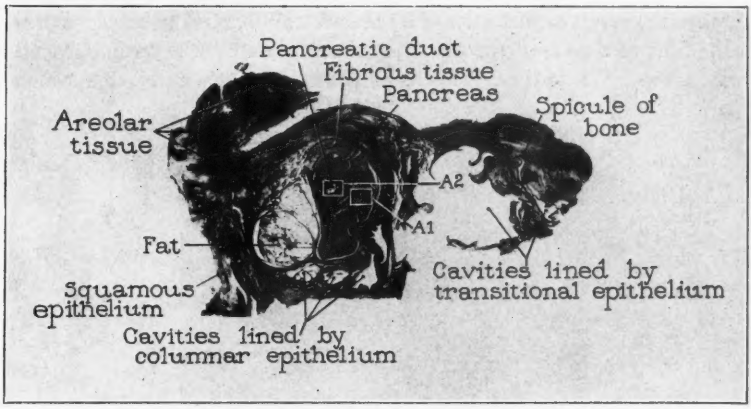
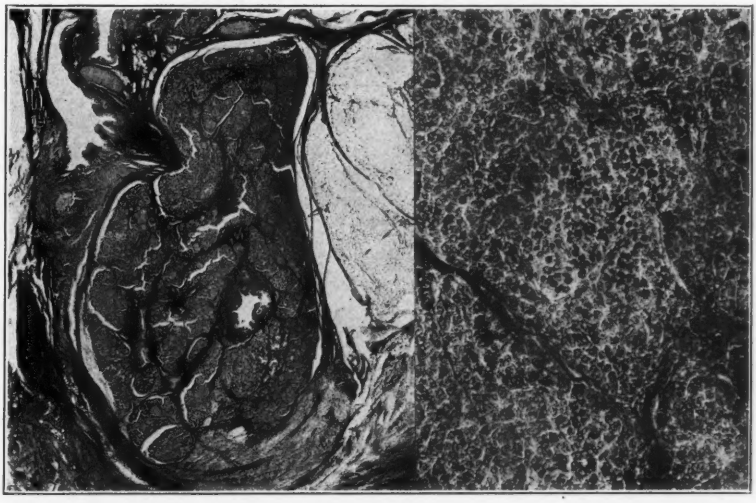


Fig. 7.—Cross section from anterior portion of parasitic fetus.



A *B*

Fig. 8 *A*.—Higher power of area of tissue from anterior portion of parasitic fetus shown in Fig. 7, containing a large mass of circumscribed pancreatic tissue. One duct and many islets of Langerhans are shown, surrounded by cavities lined by columnar epithelium and containing mucous glands which suggest rudimentary intestinal tract, also fat, areola and fibrous connective tissue; *B*, higher power of area *A1* (Fig. 7). Islet of Langerhans surrounded by pancreatic acini is shown.

and attached only to the posterior surface of the sternum. Nor have I been able to find a report of any case of a tumor containing fetal tissue in which normal-appearing pancreatic tissue with islands of Langerhans has been found.

Many hypotheses have been advanced to explain the origin of these teratoid growths. The earlier hypotheses were based on the conception that these growths are composed of tegumental structures only, and that they are the result of inclusion of an invaginated portion of the ectoderm at the time of closure of the primitive thoracic wall. The term, dermoid tumors, was given to these growths on the basis of this early view which

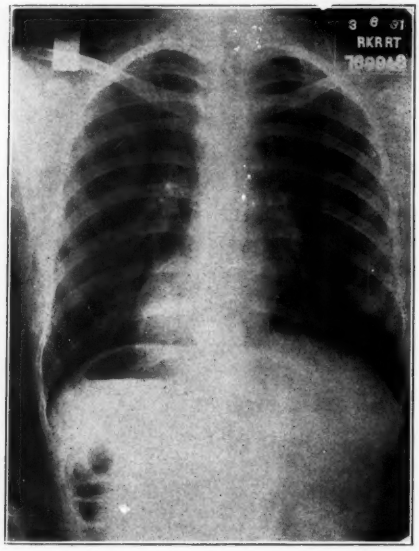


Fig. 9.—Roentgen ray taken five weeks after complete removal of anterior mediastinal tumor; mediastinum, lung and heart appear normal.

in most instances is a misnomer as the tumors are usually of more complex structure. When the histologic structure of these growths became more definitely known it was found that many contained tissue derived from the entoderm and mesoderm, and it was impossible to explain the presence of such tissue on the basis of invagination of the ectoderm. The hypothesis of origin from the thymic and thyroid glands, the branchial cleft, and the bronchi was then advanced but this failed to explain the presence of nervous tissue and mature organoid structures that were often found. Because of the presence of derivatives from all the blastodermal layers in many growths the bigeminal theory of fetus in fetu was advanced to explain the ontogenesis of the majority of these conditions. There are many examples of growths containing fetal

tissue but it is extremely rare for these growths to become so highly developed as to be recognized as a fetus. Some embryologists claim that such growths are never enclosed within the body cavity and that essentially they should exhibit traces of vertebral segments to prove the presence of metamere structure before they can be considered as bigeminal twin parasites. Histologic study in the case reported here disclosed tissue derived from all the blastodermal layers, arranged in such a high degree of organoid structure as to represent intestinal tract, spinal column, cloaca and pancreas. I believe, therefore, that this growth should be classified as a fetal parasite.

SUBCOSTAL EXTRAPLEURAL COMPRESSION OF THE LUNG*

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EXTRAPLEURAL compression of portions of the lung by packs of paraffin mixtures, gauze, rubber balloons, etc., is being used extensively in the treatment of circumscript tuberculosis, abscess, and bronchiectasis. The pack is introduced either to effect permanent compression or temporarily to produce an obliterative pleurisy preparatory to drainage operations or resections of portions of the lung. The operation may be older than we think. Scherer[1] in a discussion before the Medical Society of Magdeburg stated that Hippocrates had inserted a pig's bladder into the chest through a thoracotomy opening and inflated it.

Extrapleural compression has the advantage over thoracoplasty in keeping the bony rib cage more or less intact. Brunner[2] has called attention to this fundamental difference between the two types of operation: extrapleural compression preserves the mechanism of expectoration, while in extensive thoracoplasty looseness of the freshly resected ribs impairs this mechanism. It is especially important that expectoration be preserved in patients with large amounts of sputum, more important even in basal pulmonary suppurations than in apical ones. Any impairment of the mechanism of expectoration, even so fleeting a disturbance as administration of morphine or codeine, may be fatal to patients with large bronchiectatic cavities at the base of the lung.

Different modes of access to the chest have been chosen with the object of preserving the integrity of the rib cage so that the various expiratory forces that combine to make a cough may not be robbed of their support.

The original method of Baer[3] who made an anterior incision and resected the second rib was modified by Stöcklin[4] who advised severing the rib, displacing its lateral portion upwards or downwards without resecting it, and reuniting the severed portions. Sauerbruch[5, 6] resects a portion of the third rib posteriorly, relying on the heavy scapular musculature for support. Krampf,[7] of the Sauerbruch clinic, advises a form of temporary rib resection in which a beveled section of one or more ribs is removed, the pleura peeled off from the endothoracic fascia, paraffin introduced into the resulting cavity, and the sections of rib replaced, being held in position by sutures passed through drill holes.

These procedures have seemed unnecessarily complicated.

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*From the Division of Surgery, Stanford University Medical School, San Francisco, California.
Read at the Fourteenth Annual Meeting of the American Association for Thoracic Surgery at San Francisco, July 1, 2, 3, 1931.

In eleven operations for paraffin compression of the apex and in several in which a large area of a lower lobe was compressed with several yards of gauze preparatory to cautery pneumotomy the ribs were not resected at all, but were freed of their intercostal musculature and the pleura pushed away from under them.

This procedure is probably not new. Sauerbruch[5] mentions it in a footnote saying that it was recommended by Ziegler. A diligent search of Ziegler's writings, however, fails to find references to the method. At any rate Sauerbruch condemns it, saying that access through the

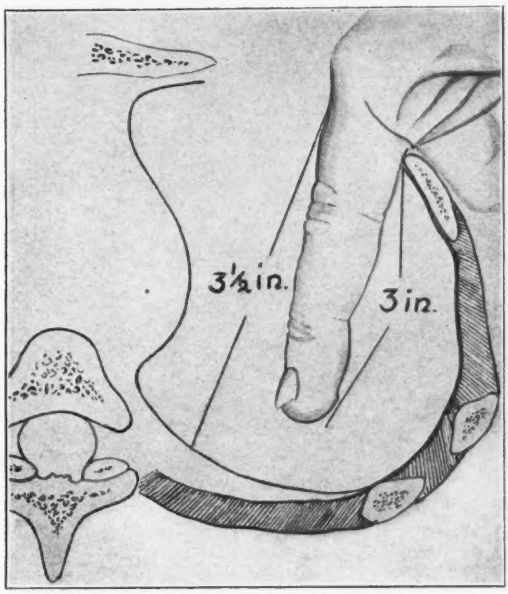

Fig. 1.—Transverse section of apex of chest showing area accessible to index finger introduced between the first and second ribs.

intercostal spaces is insufficient for the introduction of large paraffin packs and that dissection of the lung from the chest wall lacks clearness unless a rib is resected. Whether subcostal compression is done or whether a rib is severed, sprung aside, and later fastened back in place, may not be essential in apical compression, but preservation of the intact rib cage is important in bronchiectases and may be vital in patients with large amounts of purulent sputum.

Two supplementary apicolyses and paraffin compressions performed during the past year had been preceded first by a posterior then an anterior thoracoplasty. In both wax was introduced through an axillary incision; in both an exudate formed which was absorbed spon-

taneously in the one, but needed aspiration in the other. Three subcostal paraffin compressions healed without the least disturbance.

Through the kindness of Dr. Berger, of the San Francisco Coroner's Office, I was permitted to try subcostal pneumolysis on the cadaver and made measurements of the cavity. The thorax of a robust male cadaver at the level of the second rib measured 3½ inches in its greatest diameter. I was able to introduce my index finger into the first intercostal space for a distance of 3 inches. In operations on the living it has been necessary at times to use gauze mounted in a sponge stick to separate the

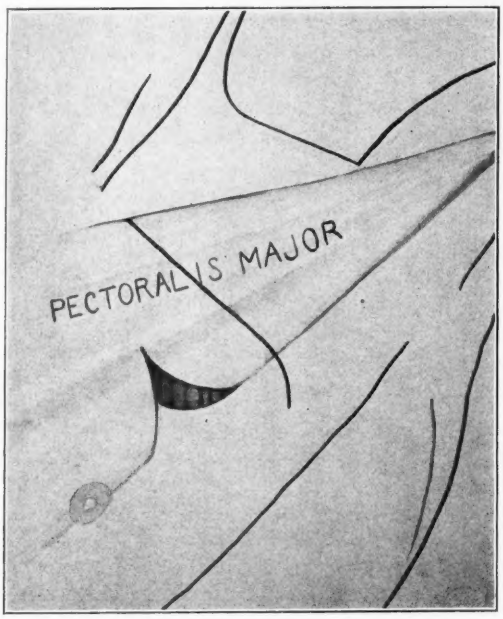

Fig. 2.—Incision for apical compression.

dome of a lung which was adherent over an area greater than my finger could reach; ordinarily the finger has sufficed.

The technic is as follows: The patient lies on his back with the arm abducted 90°. An incision is made transversely across the course of the great pectoral muscle extending 1½ inches beyond its lower border toward the axilla. Skin and fascia are dissected back an inch or two where they adhere to the lower border of the muscle. If access is insufficient, the lower edge of the muscle may be incised, the incision being made close enough to the sternum to avoid injuring the nerve and vessels which enter from underneath. The muscle is retracted up and outward. The upper and lower borders of the second rib are carefully freed of their intercostal tendons with a knife and the pleuroperiosteal layer is

peeled off the bone, dissection being carried upward, medially, and backward until the dome of the lung is well freed. An occasional adhesive strand may require ligation. If access through one intercostal space is insufficient, the third rib may be freed, so that one may work through both the first and second intercostal spaces.

The paraffin mixture is moulded into rolls about the size and shape of a large finger, introduced between the ribs and pressed into all the crevices of the subcostal cavity until the whole cavity is filled up to the level of the ribs. Sauerbruch's posterior route may be better for large

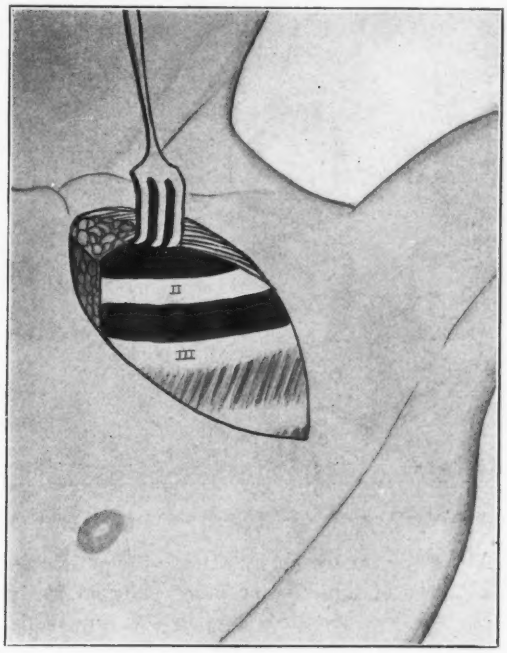

Fig. 3.—Apical compression; first and second intercostal spaces freed.

posterior cavities; it has the disadvantage of necessitating a much longer, bloodier incision, but it gives better access to the posterior and especially the medial parts of the lung. Posterior adhesions are likely to be firmer than anterior ones. The posterior incision therefore may be less likely to rupture an anterior cavity and may permit of more thorough investigation of a densely adherent apex. The subcostal anterior route is easier, less bloody, and less destructive.

Several recent publications from Sauerbruch's[8] clinic recommend extrapleural compression with wax in the treatment of abscess and bronchiectasis. At times compression is sufficient to accomplish oblitera-

tion of the abscess cavity, at others the abscess may break through into the wax-containing space. In either instance an adhesive pleurisy is produced. Gauze accomplishes the same result. In such operations where wide areas of the lower parts of the lung are to be compressed and the pleural space obliterated, it is essential that the integrity of the rib cage be preserved. The thicker muscles, the shorter and less mobile ribs of the upper part of the chest allow of rib resection without greatly impairing stability; resection of considerable areas of two or more of the lower ribs makes respiration painful and cough impossible unless the

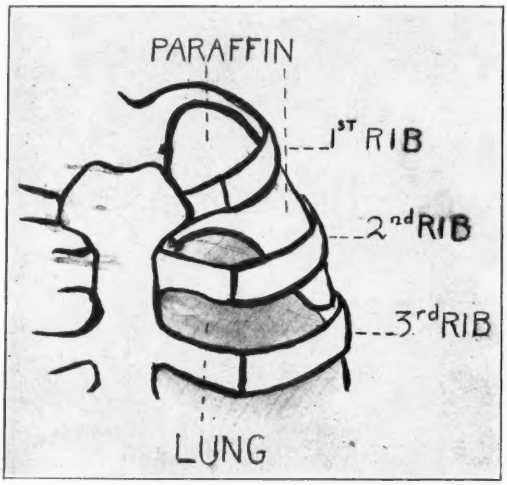

Fig. 4.—Apical compression. Diagram of rib cage showing position of paraffin mixture.

chest is immobilized by sand bags, by placing the patient on the affected side, by thick layers of adhesive or other external splints. A bronchiectatic nurse who was operated upon in October, 1928, died after a simple rib resection and extrapleural packing before the intended cavity operation could be performed, doubtless from difficulties of expectoration. In such patients subcostal compression offers great advantages without the drawbacks of lack of room and inaccessibility inherent to a similar procedure at the apex. There is unlimited room; one, two, or three intercostal spaces may be opened and the pleura peeled away from the ribs over an unlimited area, both in front and in back. There is little or no pain after operation, no painful cracking back and forth of rib implants, and no interference with expectoration. If after operation it should seem wise to abandon the plan of a later pneumotomy or lobectomy, then the wound may be reopened, the foreign material (wax or gauze) removed from under the ribs, the pleura allowed to re-expand and the skin closed as before, without harm being done; if the

planned second stage is to be carried out, to cut through the sections of rib which has been bared at the first stage is but a matter of a few seconds. This plan of deferring section of the ribs to the second stage and merely skeletonizing them at the first one is obviously simpler and easier than temporary resection with reimplantation.

Summary.—A method of subcostal extrapleural pneumolysis is described and its use recommended. It is less essential in compression of the apex, but especially advantageous in compression of the lower parts of the lung.

REFERENCES

1. Scherer: Zentralbl. f. d. ges. Tuberk.-Forsch. **22**: 353, 1924.
2. Brunner: Die chirurg. Behandl. d. Lungentbc., Leipzig, 1924, p. 284.
3. Baer, G.: Die extrapleurale Plombierung bei Lungentbc. München med. Wchnschr. **68**: 1582, 1921.
4. Stöcklin: Behandlung. d. Lungentbc. mit Pneumolyse u. Paraffin Plombierung. Ztschr. f. Tuberk. **35**: 241, 1922.
5. Sauerbruch: Chirurg. d. Brustorgane, ed. 3, Berlin, 1930, Vol. I, p. 1093 et seq.
6. Nissen: Surgical Treatment of Tuberculous Cavities of Lungs With Filling "Plombe," Surg. Gynec. & Obst. **52**: 732, 1931.
7. Krampf: Die Bronchiectasen Krankheit., Ergebn. d. Chir. u. Orthop. **23**: 639.
8. Sauerbruch: loc. cit. **1**: p. 843.

PHRENICECTOMY FORCEPS*

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HOLDEN, MASS.

THE forceps commonly used in avulsing the nerve, in the operation of phrenicectomy, obtainable from instrument dealers under various names, such as Thiersch's, Proctor's, etc., have contributed to smoothness of technic. However, in any that we have been able to secure from the instrument houses, heretofore, the serrations on the faces of the blades have extended crosswise. On two occasions in our experience, the

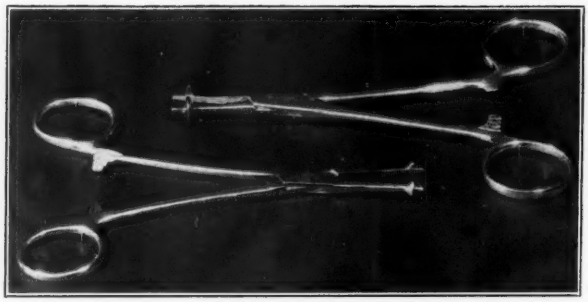

Fig. 1.

nerve has slipped from the forceps, after cutting, and during the act of wrapping the nerve on the forceps. We believe this could have been avoided had the serrations extended longitudinally, and we have had some instruments made with this correction, by Sharp and Smith, of Chicago. There is a sense of greater security in their use. In observing others operate, we have noted the additional use, occasionally, of mosquito forceps to secure the grasp of the severed end of the distal portion of the nerve, which indicates that similar fault, or fear of it, has been experienced by others.

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From the Holden Clinic.

Department of Reviews and Abstracts

CONDUCTED BY DUFF S. ALLEN, M.D., ASSOCIATE EDITOR

Selected Abstracts

Morin, J., and Rautareau, R.: Résultats et indications de la phrénicectomie dans la tuberculose pulmonaire suivant l'état anatomique de la pleure. (The Indications and Results of Phrenicectomy in Pulmonary Tuberculosis According to the Anatomic State of the Pleura.) Rev. de la tubere. 12: 593, 1931.

The notably good results from phrenicectomy have been observed only when the exeresis has been done for relatively fresh lesions in which, because of pleural obliteration, pneumothorax has been unsuccessful. Chronic lesions with marked fibrotic retraction of the affected side hold forth no promise of benefit from phrenicectomy and should be subjected to thoracoplasty. If thoracoeaustic severance of adhesions is impossible, phrenicectomy may assist in furthering the collapse when stringlike adhesions prevent the success of pneumothorax. However, if wide bandlike adhesions broadly bind the lung to the parietal pleura over the lesion, phrenicectomy will have little if any effect in furthering collapse of the affected portion. These cases should be submitted to thoracoplasty after a preliminary phrenicectomy. To determine the relative merits of phrenicectomy and pneumothorax the authors performed phrenicectomy alone on a number of cases in which pneumothorax is usually considered indicated and was mechanically feasible. Their observations lead them to prefer pneumothorax particularly where the lesions are widespread. They emphasize that good effects from phrenicectomy may be expected only if the lesion be relatively restricted and relatively fresh and the pleural space over the affected site be relatively free.

Mues, H.: Isolierte Phrenicusexairese bei Lungentuberkulose. (Phrenicectomy as the Sole Procedure in Pulmonary Tuberculosis.) Deutsche Ztschr. f. Chir. 233: 20, 1931.

The author reports a series of 65 cases of pulmonary tuberculosis treated by phrenicectomy alone, analyzed after periods of observation varying from four to ten years. He concludes that with lower lobe processes phrenicectomy alone as a single attempt at collapsotherapy can furnish excellent results. Phrenicectomy alone in cases of upper lobe or total one-sided involvement, where other collapse measures are impossible or unfeasible, very commonly can secure good ameliorative effects. Restoration of diaphragmatic function was observed in a few cases, notably those in which but a small portion of the phrenic nerve was involved. Early and late deleterious results of phrenicectomy in this series were infrequent, fleeting and of little consequence.

Neddermeyer, A., and Walther, G.: Beobachtung bei Phrenicexairese (Experience with Phrenicexeresis). München. med. Wchnschr. 78: 476, March, 1931.

The authors report their experience with 100 cases of phrenicectomy performed since October, 1928. In 80 per cent of their cases cavities were present. Unilateral

lesions occurred in only 33 per cent of the cases. Twelve of the cases had artificial pneumothorax antecedent to phrenicectomy, 95 per cent of the cases had tubercle bacilli in the sputum.

The results are summarized as follows: (1) After operation the diaphragm ascended to the VI space in 30 cases, to the V space in 46 cases and to the IV space in 20 cases. (2) The lesions of the side operated upon were improved clinically and radiologically in 77 per cent of the cases; whereas those of the contralateral side were improved in 26 per cent of the cases. The modifications undergone by the fever, the expectoration, the weight and the general clinical picture are closely commensurate with the localization of the lesion and the degree of ascent of the diaphragm.

Sorrel, E., and Bufnoir, P.: Osteite Costale avec fracture spontanee. (Costal Osteitis with Spontaneous Fracture.) Rev. d' Orth. et de Chir. de l'Appareil moteur. XXXVIII, XVIII, 3. P. 232, 1931.

A case in a boy of four of tuberculous costal osteitis of the fifth right rib with spontaneous fracture posteriorly is reported. A large thoracic wall abscess was present, which led one to consider Pott's disease. Lipiodol injection into the abscess demonstrated its costal origin. The following operative technic has been found to be very satisfactory for many years; incision into the abscess, search for the osseous origin of the disease, costal resection, total debridement of the extension of the disease below and behind the rib and extirpation of the wall of the abscess. Another method which is to be highly recommended in theory is the ablation "en masse" of the entire abscess with the affected rib without opening of the abscess, but this is difficult to put into practice. Moreover this latter procedure has no actual superiority in that it is the rule after the former procedure to obtain primary union after primary suture if fistulization has not been present and if complete debridement has been performed.

The authors point out (1) the utility of lipoidol injection in cases of cold abscess to determine the origin of the tuberculous suppuration, (2) the extreme rarity of spontaneous fracture in tuberculous osteitis of the rib (only one instance in 48 cases of tuberculous costal osteitis operated by Sorrel at Berck Sur-Mer), (3) the absence of pleural lesions in tuberculous costal osteitis in children, (4) the normal repair of the rib after resection and debridement in tuberculous costal osteitis in children.

Perry, I. H.: Experimental Study of the Inhalation of Fluid During the First Respirations. Anat. Rec. **50**: 303, 1931.

In a recent analysis of 800 autopsies of infants dying within the first month of life in Glasgow the high incidence of 32.5 per cent classified as due to asphyxia challenges more detailed consideration of the phenomena attendant upon the first respirations.

This study is an experimental investigation of the significance of amniotic fluid inhaled into the lungs of the newly born. Investigation was directed to ascertain (1) how the fluid enters the respiratory tract (2) in what amounts (3) its lethal nature. Pregnant guinea pigs were used in all the experiments. Potassium ferricyanide was injected into the amniotic sacs of the young guinea pigs after laparatomy and exposure of the uterus. Delivery of the pigs was then performed by cesarean section at stated intervals. After delivery and death of the young, the thoracic contents were eviscerated and transferred to ferric chloride solution. This solution combined with the ferrocyanide when present to give prussian blue. To further observe the position of the fetus and the number and character of the respirations the guinea pigs were also delivered by cesarean section when in a bath of ferrocyanide.

Amniotic fluid is not a constant finding in the lungs of the human or guinea pig young. However when present the mode of entrance of amniotic fluid is not by gravity which presupposes an upright position and an open larynx. The human fetus is most frequently head down during delivery. The experiments indicate that premature young are capable of aspirating fluid into the lungs against gravity. The inhaled fluid is seldom sufficient in bulk to be the cause of the asphyxia by obstruction of the bronchi and alveoli. The amount of inhaled fluid is usually small compared with the great reserve of pulmonary surface area. Inhalation of fluid is not incompatible with viability. The conclusion is drawn that the finding of inhaled fluid within the lungs of the newborn is usually an indication of asphyxia rather than a cause of it.

Denéchan, N., and Fruchard, H.: Pyothorax fistulisé consécutif à un pneumothorax thérapeutique. Thoracoplastic élargie. Résection des apophyses transverses. Résection de la moitié inférieure de l'omoplate. Pleurectomie étendu. Guérison. (Empyema With Bronchopleural Fistula Secondary to Therapeutic Pneumothorax. Extensive Thoracoplasty. Resection of Transverse Processes. Resection of Inferior Portion of Scapula. Extensive Pleurectomy. Cure.) Rev. de la tuberc. **13**: 173, 1932.

The report concerns itself with the treatment of a case of secondarily infected (pneumococcus) tuberculous empyema subsequent to artificial pneumothorax treatment. The following consecutive procedures were carried out (1) drainage, (2) oleothorax, (3) paravertebral thoracoplasty in two stages, (4) resection of the lower position of the scapula to efface a persistent infected residual cavity, (5) revision of the thoracoplasty with further resection of the stumps of the previously resected ribs and resection of the transverse processes, (6) extensive pleurectomy. An excellent result was obtained with return to occupation. Moderate deformity is present, but good function of the arm and shoulder is present.

The author comments on the treatment of tuberculous pyothorax, secondarily infected, of long standing with bronchopleural fistulae. Lavage of the pleura with antiseptic solutions has proved useless in his hands. Only in mildly infected, recent cases is pleural lavage of any avail and then only when associated with pleurotomy. Furthermore "medical" treatment demands long periods of time and permits infection to penetrate deeply into the tissues and render the pleura a stiffened rigid shell. Furthermore, one should not forget that behind the infected pleura is a tuberculous lung which requires collapsotherapy to cure the underlying disease. Collapsing operations in cases of tuberculous empyema have not nearly the gravity that one has been led to believe. Because of the thickening of the mediastinal pleural lamellae strong mediastinal fixation is usually present. Acute cardiorespiratory deaths due to mediastinal flutter, such as not infrequently attend thoracoplasty, are rare after thoracoplasty for tuberculous empyema, and the patients in spite of their exhaustion from fever and toxicity withstand the operation remarkably well. In cases with supple thoracic cages in which the pleura is not greatly thickened, extrapleural thoracoplasty usually suffices. More advanced cases with thicker pleura require anterolateral costectomy in addition. In long standing cases with rigid thickened pleura pleurectomy is necessary. Bernou has reported excellent results in this type of case with pleurothoracectomy. The operation should be done in several stages to avert shock. If left to themselves these patients usually succumb to the inexorable march of sepsis. Excellent therapeutic results can be obtained in some cases if surgical collapsotherapy is employed.

Book Review

SURGERY OF THE CHEST, by George F. Straub, M.D., F.A.C.S., with three hundred and forty-one illustrations including sixty-eight color plates. Charles S. Thomas, Publishers, 1932.

It is easier to criticize a book than to write one. A reviewer very naturally should search for the imperfections as well as for the good points. It often happens also that in a controversial matter a reviewer unjustly criticizes the author's point of view merely because he disagrees with it. With a full realization of the axioms just mentioned this reviewer has read Dr. Straub's book. Within the 475 pages the author has, on the whole, presented the operative technic of thoracic operations very well. There is a serious lack, however, of adequate discussion of the conditions themselves for which various operative procedures are described. In the reviewer's opinion this lack constitutes an important objection because already in this comparatively new field of surgery many operations are being unwisely performed by those who are not qualified either by adequate knowledge of the conditions or by a sufficiently sound training in the fundamental principles of general surgery. The comparative simplicity of the technic of many of the thoracic operations has encouraged many to plunge into this field who are utterly unqualified.

A reader who knew nothing about thoracic surgery could hardly gain a satisfactory impression of the dangers and the results to be expected from various methods of surgical attack on thoracic disease from a reading of this book. Moreover, in most of the conditions which are discussed the symptoms and diagnosis are very inadequately presented. The pathologic features receive almost no attention at all. The American literature is not so well presented as one would expect. It is difficult to understand how anyone at all familiar with the experience of the United States Army Empyema Commission, or for that matter with any of the modern writings on the subject of empyema, could advocate the treatment which the author recommends for what he considers to be the most severe form of virulent streptococcal empyema. For example, he states "these patients, if subjected to prompt operation, have a fair chance of recovery . . . . the treatment consists of early and wide opening of the pleural cavity with resection of from two to four ribs, careful exploration of the entire space, and tamponade." In the discussion of the treatment of chronic empyema no adequate presentation of the principles is given and there is no indication of the fact that many

cases of this condition are due to inadequate drainage whether from too small an opening or an improperly placed one. In the experience of most surgeons who have had to deal with many of these cases the simple use of good drainage in some cases results in a complete healing of the empyema, and in nearly all cases in which the drainage has been inadequate it results in a marked diminution in the size of the cavity so that extensive surgical measures need be less radical than they would be without this simple preliminary treatment. The author, however, does not mention this well known and simple expedient. There are many, including the reviewer, who would disagree with the statement that there is a "striking facilitation of cough after paralysis of the diaphragm" (page 190). Sometimes one sees a patient in whom the act of coughing is made easier but there are also many patients in whom it is made much more difficult. It is unfortunate to have an unqualified statement like the one just quoted appear in a book devoted to surgery of the chest because it is misleading.

The illustrations for the most part are diagrams made by the author. For several of the illustrations the plan used by Sauerbruch in his book has been adopted; the muscles have been printed in red to distinguish them from other tissues.

Most of the descriptions of operations are well done. The chapter entitled "Glimpses Into the History of Thoracic Surgery" is a very interesting one.

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In Memoriam

SELLING BRILL, M.D.

(DECEMBER 27, 1899—JANUARY 23, 1932)

A CAREER of remarkable promise in experimental and clinical surgery was unfortunately and unexpectedly terminated by the death of Dr. Selling Brill as a result of bacterial endocarditis. His loss is a grievous one to the University of California Medical School and it is especially distressing to those of his colleagues who had the pleasure of working with him.

Dr. Brill came to this country with his family from Russia in 1906. Since his mother died when he was quite young, particularly affectionate were the ties which bound him to his father and brother, both of whom survive him. His brother, Dr. I. C. Brill, of Portland, Oregon, brought him to San Francisco in 1914 so that he might be fitted to enter Stanford University.

Under the guidance of his brother he prepared for a medical career following his graduation from Stanford in 1920. After a year at Har-

vard Medical School he returned to Stanford to engage in a year's research effort under Professor W. H. Manwaring on hepatic reactions in anaphylaxis, for which he was awarded a Master's degree in 1922. He also won academic honor by election to Phi Beta Kappa and Sigma Xi. He took his doctorate in medicine at Harvard in 1924 and then spent two years as a house officer on the staff of Dr. Harvey Cushing at the Peter Bent Brigham Hospital. From Boston Dr. Brill went to the University of Pennsylvania where he obtained a Fellowship with Dr. Charles Frazier and Dr. I. S. Ravdin on the surgical staff of the University Hospital. Here he engaged in experimental studies on water metabolism and intraperitoneal temperature, and carried through an important clinical investigation of intestinal obstruction.

In 1929, upon the establishment of the J. J. and Nettie Mack Foundation in connection with the George and Irene Roos Clinic on Thoracic Surgery at the University of California Medical School, Dr. Brill was persuaded to assume responsibility for the experimental studies associated with the work of the clinic. With characteristic skill and vigor, Dr. Brill undertook a series of fundamental investigations concerned with factors involved in massive collapse of the lung and with alterations of intrapleural pressure. He also studied the effects of various surgical manipulations on the functional activity of the lungs. His generous enthusiasm in this work stimulated all those associated with him, and attracted the interest of many brilliant pupils. These studies developed into an investigation of the etiology of emphysema and led to many important applications which have received wide attention and commendation.

In the brief span of Dr. Brill's professional career he made a great number of important contributions to experimental surgery. He won the respect and admiration of all who knew him. He was an inspiring teacher and preceptor to those who sat under him for instruction. His unfailing courtesy and his critical acumen endeared him to his professional colleagues and it is with the utmost sorrow that they have witnessed his passing from their midst.

The following is a list of Dr. Brill's publications:

1. Brill, S., and Manwaring, W. H.: Hepatic Reactions in Anaphylaxis; Vasomotor Reactions in Isolated Canine Liver, J. Immunol. **8**: 47-53, 1923.
2. Brill, S., Boyd, W. H., and Manwaring, W. H.: Hepatic Reactions in Anaphylaxis; Hepatic Mechanical Factor in Peptone Shock, J. Immunol. **8**: 121-130, 1923.
3. Brill, S., Manwaring, W. H., and French, W. O.: Hepatic Reactions in Anaphylaxis; Mechanism of Increased Hepatic Resistance During Canine Peptone Shock, J. Immunol. **8**: 211-215, 1923.
4. Brill, S., and Appel, K. E.: Postoperative Water Metabolism and Intradermal Salt Solution Test; Preliminary Report, Ann. Surg. **85**: 502-508, 1927.
5. Brill, S., and Muller, G. P.: Gastrostomy in Carcinoma of the Oesophagus, Ann. Surg. **88**: 48-57, 1928.
6. Brill, S.: Glycogenolysis Due to Epinephrine in Hepatic Disease, Arch. Surg. **18**: 1803-1816, 1929.

7. Brill, S.: Mortality of Intestinal Obstruction: Analysis of 124 Cases Operated Upon at the Hospital of the University of Pennsylvania, Ann. Surg. **89**: 541-548, 1929.
8. Brill, S.: Effect of Abdominal Thermal Application on Intraperitoneal Temperature, Ann. Surg. **89**: 857-866, 1929.
9. Brill, S.: Effect of Sodium Amytal on Hyperglycemia Due to Morphine, Proc. Soc. Exper. Med. & Biol. **27**: 265-267, 1930.
10. Brill, S., and Leake, C. D.: Effect of Typical Broncho-Dilating Drugs on Intrapleural Pressure, Proc. Soc. Exper. Biol. & Med. **27**: 518-521, 1930.
11. Brill, S., and Lawrence, L. B.: Changes in Temperature of Lower Extremities Following Induction of Spinal Anesthesia, Proc. Soc. Exper. Biol. & Med. **27**: 728-731, 1930.
12. Brill, S., and Leake, C. D.: Experimental Studies on Factors Altering Intrapleural Pressure, Am. J. Physiol. (Proc.) **93**: 636-637, 1930.
13. Brunn, H., and Brill, S.: Atelectasis: a Review of Its History, Significance, and Treatment, West. J. Surg. **38**: 647-662, 1930.
14. Brunn, H., and Brill, S.: Observations on Postoperative Pulmonary Atelectasis; Consideration of Some Factors in Its Etiology, Prevention, and Treatment, Ann. Surg. **92**: 801-837, 1930. Also in Trans. Am. Surg. **48**: 53-59, 1930.
15. Brill, S., and Brown, A. L.: Experimental Production of Mucous Plugs in Bronchi of Dogs, Proc. Soc. Exper. Biol. & Med. **28**: 533-535, 1931.
16. Brill, S., Prinzmetal, M., and Leake, C. D.: Effects of Certain Broncho-Constricting Drugs on Intrapleural Pressure, Proc. Soc. Exper. Biol. & Med. **28**: 617-620, 1931.
17. Brill, S., Lawrence, L. B., and Naffziger, H. C.: Temperature Rise in Lower Extremities Following Spinal Anesthesia; an Aid in Selection of Cases for Lumbar Sympathectomy, West. J. Surg. **39**: 180-191, 1931.
18. Prinzmetal, M., Brill, S., and Leake, C. D.: Effects of Carbon-Dioxide Inhalations on Intrapleural Pressure in Dogs, Proc. Soc. Exper. Biol. & Med. **28**: 832-835, 1931.
19. Prinzmetal, M., Brill, S., and Leake, C. D.: Changes in Thoracic Size Following Administration of Broncho-Constricting or Broncho-Dilating Drugs to Dogs, Proc. Soc. Exper. Biol. & Med. **28**: 836-838, 1931.
20. Prinzmetal, M., Lonergan, L., and Brill, S.: Effects of Anoxemia on Intrapleural Pressure in Dogs, Proc. Soc. Exper. Biol. & Med. **29**: 191-193, 1931.

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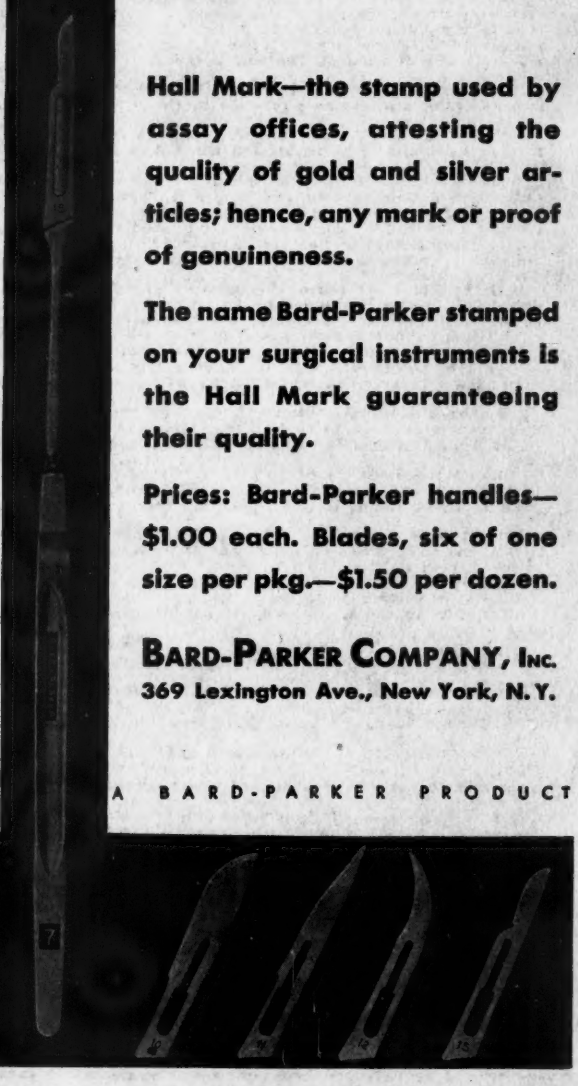

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